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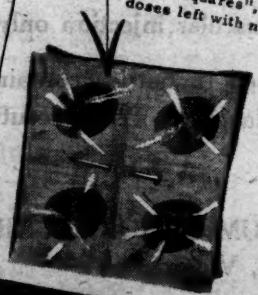

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SUBARACHNOID HÆMORRHAGE.[1]

By J. G. RADFORD,
Sydney.

TONIGHT it is my privilege to read the first of three papers on the subject of subarachnoid hæmorrhage. These three papers may be described in a sense as representing the past, the present and the future. I shall endeavour to recapitulate the purely clinical aspect of the picture, which has not changed much since the past. Dr. Burke will describe to you the present contribution which modern diagnostic aids can make, and Dr. Morson will describe the surgical attack on this problem, which will become more and more important in the future. Most of my remarks will contain nothing new, but I shall attempt to refresh in your minds the clinical picture, drawing partly on my own experience, partly on the writings of others, and partly on a review of 50 cases extracted from the records of the Royal Prince Alfred Hospital.

Definition and Ætiology.

The term subarachnoid hæmorrhage requires no definition, since it implies in its literal sense bleeding into the subarachnoid space of the meninges. This can occur in a number of conditions. Tonight we are chiefly concerned with subarachnoid hæmorrhage due to rupture of an aneurysm of one of the vessels of the circle of Willis or its tributaries, the condition most commonly envisaged when the term subarachnoid hæmorrhage is used. Certain other conditions must be mentioned which may lead to the presence of blood in the subarachnoid space, and which must be differentiated from "Willisian" hæmorrhage.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 27, 1954.

Subarachnoid Hæmorrhage Secondary to Cerebral Hæmorrhage.

Blood from one of the main vessels on the surface or in the substance of the brain may extravasate into the subarachnoid space by hæmorrhage out to the surface or down into the ventricles and thence into the subarachnoid space. The distinction of this condition from berry aneurysm may at times be difficult. Hypertension is usually present and hemiplegia also, but neither of these rules out primary subarachnoid hæmorrhage. As regards the relevant frequency of these two conditions, in 50 cases at Royal Prince Alfred Hospital classified as "intracranial hæmorrhage, non-traumatic", 21 were classified as due to subarachnoid hæmorrhage, 26 to ordinary cerebral hæmorrhage, one to cerebellar hæmorrhage, and one to subdural hæmatoma, and there was one readmission.

Angiomatous Malformations.

I refer to angiomatous malformations next, because, although they are uncommon, they are being more frequently recognized and may be amenable to surgical treatment. Their existence may be suspected by the

occurrence of focal epilepsy and monoplegia. Outward and visible associated angiomata may be seen on the surface of the face or scalp of the same side (Sturge-Weber-Dmitri syndrome). Hæmaturia from associated renal angiomata may occur in the Lindau syndrome.

Trauma.

Trauma must be carefully excluded.

Encephalitis and Meningitis.

Severe encephalitis and meningitis may rarely be associated with bleeding into the subarachnoid space. I mention this because I have seen meningitis elude diagnosis when, in a case in which neck stiffness and coma were present, the attending physician was led astray by the obtaining of blood-stained fluid on lumbar puncture and the cerebro-spinal fluid was never cultured for organisms.

Mycotic Aneurysm.

Mycotic aneurysm should not be forgotten, and the presence of subacute bacterial endocarditis should be excluded.

Blood Dyscrasias.

Purpura and other blood dyscrasias will occasionally cause subarachnoid hæmorrhage. The first case of subarachnoid hæmorrhage which I encountered in my student days was in a young girl with idiopathic thrombocytopenic purpura.

Venous Hæmorrhage in Pyæmia.

Venous hæmorrhage in pyæmia must surely be unusual in these days.

Syphilis.

Syphilis, according to Russell Brain (1947) is an unlikely cause of cerebral aneurysm, because in small arteries it produces an obliterating endarteritis. However, the first case in one of the Royal Prince Alfred Hospital series was one of syphilitic aneurysm.

Pathogenesis of Aneurysms of the Circle of Willis.

I want to mention briefly the question of pathology. The occurrence of aneurysms on or about the circle of Willis has been ascribed to a congenital deficiency at or near the bifurcation of a cerebral artery. There is absence of the elastic and muscular elements, and there is a narrow fibrous media. The aneurysms may vary from the size of a pin's head to 30 millimetres. They may be single or multiple. It is alleged that they develop at any stage of life, and Brain (1947) states that 80% rupture sooner or later. This may occur at any age; it happens most commonly between forty and fifty years, but it also occurs in youth or old age, and more commonly in females than in males. Support for the congenital origin of this condition is derived from the pathology of the condition and the occasional association with other congenital anomalies, for example, abnormalities of the abdominal vessels, coarctation of the aorta and polycystic disease of the kidneys (Poutasse *et alii*, 1954). Bigelow (1953) in 8892 autopsies at the Albany Hospital found 47 cases of berry aneurysm, and in three of these subjects polycystic kidneys were also present (6·4%); he also found 18 subjects with polycystic kidneys, of whom three had intracranial aneurysms (16·6%). These figures are similar to those of Brown (1951)—namely, 4% and 16·6% respectively. Occasionally a family history is present. There is a school of thought that development of berry aneurysms is due to acquired degenerative disease of the vessels, and that hypertension is a predisposing factor in rupture. To those of us who have seen young normotensive nurses and medical students stricken with this tragic complaint the congenital hypothesis is more attractive.

Symptoms and Signs.

Symptoms before Rupture.

I wish now to refer briefly to symptoms which may appear before rupture of an aneurysm—that is, before subarachnoid hæmorrhage actually occurs. Usually there are no antecedent symptoms. However, headache has been described in 25% of cases, and in 50% of these it may be migrainous in character. Other symptoms may be due to focal pressure. These are usually sharply localized and slowly or not at all progressive. The aneurysm may cause pressure on the optic nerve with impairment of vision, perhaps transitory, perhaps with optic atrophy, rarely with papillœdema or exophthalmos. In involvement of the optic tracts hemianopsia may be present. Third, fourth and sixth nerve palsies may occur. Occasionally involvement of the ophthalmic division of the fifth nerve leads to pain or anæsthesia over its distribution. In involvement of the middle cerebral artery, monoplegia, hemiplegia or convulsions may occur. In involvement of the posterior communicating artery third nerve palsy and hemianopsia may be present. In involvement of the basilar artery crossed hemiplegia and ipsilateral cranial nerve lesions may occur. In the presence of any of these signs the possibility of the existence of aneurysm should be borne in mind, and an accurate diagnosis may enable surgical treatment to prevent the almost inevitable catastrophe.

Symptoms and Signs following Rupture.

The onset of hæmorrhage is usually sudden. Coma and fatal termination may be rapid. More usually there is the characteristic sudden throbbing occipital headache with the sensation of "something having snapped". Vomiting is frequent, photophobia is not uncommon. Neck stiffness is almost invariably present. These are all the symptoms of sudden meningeal irritation by bleeding. The signs are those due to rise in pressure and those due to focal damage.

General Signs.—The general signs include coma, generalized epileptiform convulsions, diminution of reflexes and "upgoing toes". Papillœdema may be present. Retinal and subhyaloid hæmorrhages may be seen occasionally if sought. This is thought to be due either to seepage of blood along the prolongation of the subarachnoid space onto the optic nerves or to pressure on the retinal veins. Glycosuria may be present, and as in other cerebral conditions should not lead to an erroneous diagnosis of diabetes.

Focal Signs.—Focal signs are due either to compression of nerves by clot or to invasion of the cerebrum by hæmorrhage. They may be difficult to elicit because of drowsiness or coma, but should be repeatedly sought as the patient's condition varies. Most of them have already been mentioned in the section on symptoms and signs prior to rupture. They include defects of vision, ophthalmoplegia, and fifth nerve involvement, which are examples of nerve damage. Cerebral damage may lead to mental changes, including confusional states, mania and personality changes, which may be erroneously interpreted as due to psychosis, particularly if the patient has not been examined until after recovery from the more obvious signs of hæmorrhage. Damage to the motor areas may lead to aphasia, convulsions, monoplegia and hemiplegia. Pyramidal involvement may lead to quadriplegia.

Prognosis.

It is generally recognized that approximately one-third of these patients die in the first attack. Walton (1952), in a follow-up of 312 patients, was able to trace 170 of the 172 survivors. Recurrent subarachnoid hæmorrhage had occurred in 40 (23·5%) with a fatal outcome in 35 (20·5%); half of the fatal recurrences took place within six months. Of those cases that are fatal during the first attack, death frequently occurs in the second week after a preliminary partial recovery. It is imperative, therefore, that if it is at all possible, accurate diagnosis be made early, so that surgical aid can at least be given serious consideration.

It is of interest that in spite of this gloomy outlook Brain (1947) described a case in which the patient had his first hæmorrhage at the age of sixty years, and four more before reaching the age of seventy years, at which he was still alive.

Report on Case Records at Royal Prince Alfred Hospital.

I decided to investigate the records at the Royal Prince Alfred Hospital with the object of determining how some

of our figures corresponded with those already published. I felt that clinical observation was at least as accurate fifteen years ago as it is today, and decided to go through the consecutive records of subarachnoid hæmorrhage filed in the past. My first difficulty was that at that stage we had no actual classification of "subarachnoid hæmorrhage" in our record system. I therefore perused the volume headed "hæmorrhage, intracranial, non-traumatic". I have already mentioned that 21 out of 50 of these cases were cases of subarachnoid hæmorrhage, and the details of these cases are shown in Series 1 in Table I. I then analysed a series of "aneurysms, other than great vessels". In 50 of these cases I found 29 cases of aneurysms of the "Circle of Willis" proven at autopsy. These are shown in Series 2. I have also combined these two series to give a total of 50 cases of subarachnoid hæmorrhage. While I realize the statistical inadequacy of these figures, the results are interesting, and a few conclusions may be reasonably drawn.

TABLE I.

Observation.	Series 1. (21 Subjects.)	Series 2. (29 Subjects.)	Combined Series. (50 Subjects.)
Sex:			
Males	13	10	23 (46%)
Females	8	19	27 (54%)
Age (years):			
Males	Average 48 (18 to 68)	Average 38 (21 to 60)	18 to 68
Females	Average 52 (29 to 76)	Average 44 (9 to 64)	9 to 76
Previous history:			
Headache	6	7	13
Migraine	1	2	3
Cranial nerve palsies	1	2	3
Motor disorders	3	1	4
Mental disorders	—	1	1
Hypertension	14	10	24
Symptoms and signs:			
Headache	14 (6 coma, 1 interval)	22 (6 coma, 1 interval)	36
Vomiting	15	11	26
Convulsions	3	2	5
Coma	7	16	23
Drowsiness	6	5	11
Photophobia	4	1	5
Neck rigidity	17	8	25
Papilloedema	4	8	12
Retinal hæmorrhage	1	4	5
Field defect	0	5 (3 loss of vision, 1 homonymous hemianopsia, 1 bitemporal hemianopsia)	5
Third, fourth and sixth cranial nerve palsies	8	12	20
Other cranial nerve palsies	3 (1 fifth nerve, 2 seventh nerve)	6 (5 fifth nerve)	9
Mental change	5 (3 confusion, 1 mania, 1 delusions)	7 (5 confusion, 1 mania, 1 personality disorder)	12
Motor signs	14 (9 hemiplegia, 2 monoplegia, 3 quadriplegia)	14 (7 hemiplegia, 1 aphasia, 6 quadriplegia)	28
Death:			
Within 48 hours	2	9	11
In 2 to 14 days	3 } 5	6 } 17	9 } 22
Later	—	2	2
Recovery	16	12	28

You will note that the age of occurrence ranges from nine to seventy-six years. With regard to previous history, headache was found to have been present in 26% of the cases prior to rupture, and in 23% of these it was migrainous in character. Hypertension was found in 48% of cases. With regard to the actual hæmorrhage, headache was universal except in cases in which coma supervened rapidly. Coma was present in 46% of cases. Photophobia was present in 10% of the cases. Papilloedema was present in 24% and retinal hæmorrhage in 10%. Third, fourth and sixth nerve palsies were present in 40%. Mental changes were present in 24%. Hemiplegia, monoplegia, quadriplegia and aphasia were present in 56%.

Prognosis.

Death occurred within forty-eight hours in 22% of cases and within fourteen days in 40% of cases. The total mortality in the first attack was 44%.

Site of the Aneurysm.

In the autopsy series the aneurysm was in relation to the anterior cerebral artery in three, to the middle cerebral artery in six, to the posterior cerebral artery in six, and to the basilar artery in one, and in one case there were multiple aneurysms.

Acknowledgement.

My thanks are due to the Medical Superintendent of the Royal Prince Alfred Hospital for permission to utilize hospital records in preparing this paper.

References.

BIGELOW, N. K. (1953), "Association of Polycystic Kidneys with Intracranial Aneurysms and Other Related Diseases", *Am. J. M. Sc.*, 225: 485.

BRAIN, W. R. (1947), "Diseases of the Nervous System", Third Edition, 296.

BROWN, R. A. P. (1951), "Polycystic Disease of the Kidneys and Intracranial Aneurysms", *Glasgow M. J.*, 32: 333.

POUTASSE, E. F., *et alii* (1954), "Polycystic Kidney Disease and Intracranial Aneurysm", *J.A.M.A.*, 154: 741.

WALTON, J. N. (1952), "The Late Prognosis of Subarachnoid Hæmorrhage", *Brit. M. J.*, 2: 802.

MURINE TYPHUS IN WESTERN AUSTRALIA.

By ERIC G. SAINT, A. F. DRUMMOND and I. O. THORBURN.

From the Clinical Research Unit, Royal Perth Hospital, Perth, Western Australia.

THE confusion of thought regarding typhus fever which has existed in past decades is reflected in the continued popular use of the eponym "Brill's disease" with reference to the form of endemic typhus prevalent in Western Australia. In 1910 Brill described a sporadic "typhus-like" fever occurring in New York, differing from classical louse-borne typhus in respect of its mildness. Many years later (1934) Zinsser indicated that Brill's disease might be a form of recrudescent typhus occurring in persons of European birth who had previously suffered an attack of louse-borne typhus in Europe, a postulation confirmed by Plotz (1943), who used complement fixation tests to establish the identity of the antigen in the two diseases. Unfortunately, for many years after the appearance of Brill's first paper, all subsequent cases of non-epidemic typhus in the United States of America and elsewhere were called Brill's disease. Even Maxcy (1926), who recognized that a reservoir of Rickettsiæ in rats was responsible for these sporadic human cases, perpetuated the use of the eponym. The confusing situation was clarified by Dyer *et alii* (1931), and by Mooser, Castaneda and Zinsser (1931), who isolated the causative organism (*Rickettsia mooseri*) of murine typhus from the rat flea and from rats' brains.

The history of knowledge of rickettsial infections in Australia is well known and has been reviewed by Burnet (1942), but is worth recapitulating. Epidemic louse-borne typhus is known to have occurred ("gaol fever") in convicts aboard outward-bound transportation vessels (Cumpston and McCallum, 1927). The first comprehensive description of murine typhus came from South Australia (Hone, 1922, 1923). Wheatland (1926) described a "typhus-like fever" in farm workers in the Toowoomba district of Queensland, where a rodent plague had preceded the outbreak in the human host. On the coast of Queens-

land north of Townsville "endemic typhus" was differentiated from other obscure fevers by Langan and Mathew (1935) and by Mathew (1938), and was subsequently identified by Heaslip (1940, 1941) as scrub typhus (*tsutsugamushi*). Derrick (1937) had meanwhile described the clinical features of "Q" fever, the causative organism of which (*Coxiella burneti*) was isolated by Burnet and Freeman (1937). To this trio of rickettsioses occurring in Queensland's variegated terrain a fourth was added in 1946 by Andrew, Bonnin and Williams—a tick-borne typhus of the Rocky Mountain spotted fever group occurring on the Atherton Tableland. From these cases of "North Queensland tick typhus" a hitherto unidentified Rickettsia was isolated and studied in detail by Funder and Jackson (1946), and by Plotz, Smadel, Bennets, Reagan and Snyder (1946).

Studies on rickettsial infections in Western Australia have not been carried out extensively. A small survey on sera obtained from meat-workers in Wyndham and from city abattoir workers has so far failed to reveal evidence of past infection with "Q" fever (Drummond, 1953). However, the existence of murine typhus throughout the State has been known for many decades. The purpose of this communication is to describe the epidemiological and clinical features of murine typhus in Western Australia, where the disease has been notifiable since 1927.

Total Incidence.

From 1927 to 1952, 1332 cases have been notified (Annual Reports, Commissioner of Public Health, Western Australia, 1927-1952). There have been 50 deaths, mostly among elderly patients—an over-all mortality rate of 3·7%, but an age-specific case fatality rate in patients aged over fifty years of approximately 10%. As the march of symptoms and the appearances of the exanthem are unmistakable, and as subclinical or inapparent infections are not a feature of murine typhus, the approximate accuracy of these figures cannot be doubted.

The annual incidence since 1927 is shown in Figure I. It will be seen that prior to the recent war yearly notifications varied between 13 and 60, with a mean of 30. In 1943 no less than 123 cases were reported, and the annual incidence remained high until 1949; only since 1950 have yearly notifications fallen to pre-war levels.

It is difficult to explain this sizable increment in the number of cases notified during the latter years of the war and within the first five post-war years. During the war it is likely that expansion of the rat population occurred as a result of inadequate vermin control. Since then the activities of official rat-catchers and the pride of property owners have been reasserted, and DDT and "Chlordane" have been liberally used—measures which have presumably altered ecological balance to the detriment of rats and their ectoparasites. Whatever may be the true explanation, the decline in incidence of murine typhus has occurred proportionately in all areas of the State from which cases are notified.

Geographical Incidence.

The distribution of cases notified since 1947 is shown in Figure II. Most cases are reported from the metropolitan areas of Perth and Fremantle. Other foci of infection are present in the port of Geraldton and a few nearby wheat-growing towns, in the southern ports of Bunbury and Albany and nearby towns, and in a group of dairying towns situated on the Murray River, 40 miles south of Perth. In recent years isolated cases have been reported in the remote south-east (Lake Grace) and in the remoter north-west (Wyndham).

In the metropolitan areas most cases have occurred among workers in rat-infested city warehouses, granaries, flour mills—the disease is a legally recognized occupational risk—and in the inhabitants of old, rat-infested properties. Distribution throughout the suburbs is patchy; many cases are notified from Midland Junction, where the State's largest railway goods yard is situated, from Gosnells, a centre of market gardens and small poultry farms, and from two suburbs south and west of the River Swan (South Perth and Mosman Park).

Generally speaking, the incidence of typhus in the State follows closely the probable distribution of the rat population around centres where grain and provender are bulk handled and stored, either on a large scale (granaries, mills) or on a small scale (poultry farms, stables).

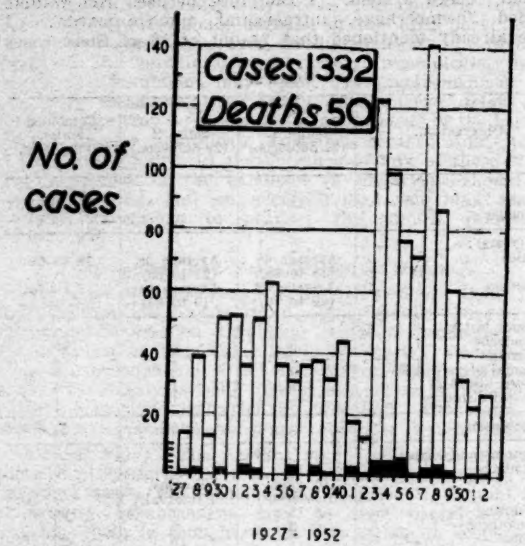

FIGURE I.
Annual incidence of typhus fever in Western Australia, 1927-1952 inclusive.

Seasonal Incidence.

The seasonal incidence of typhus in four representative years is shown in Figure III. Although cases are reported throughout the year, there is a preponderance during the months of summer and autumn.

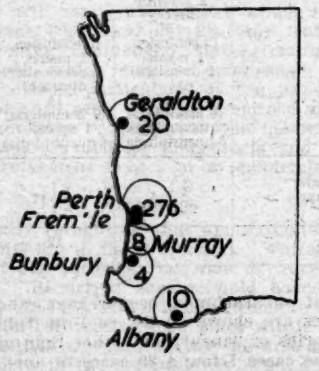

FIGURE II.
Geographical incidence of murine typhus in Western Australia.

Clinical Features.

The ensuing observations are based on information obtained from 96 patients admitted to the Royal Perth Hospital between 1944 and 1953. Only patients with a rise in titre of agglutinins against *Proteus OX19* from 0 to at least 1 in 200 were included for the purpose of this study.

The majority of patients had acquired their infection in the metropolitan area of the State. There were 49 males

and 47 females. The maximal age incidence was in the fifth decade of life (see Figure IV). The occupations of 82 of the patients are shown in Table I.

In nearly half the cases contact with rats had occurred in and around the home, and in the remainder contact had occurred at work in rat-infested warehouses, factories, eating houses, granaries, stables and fowl runs. Memory of having been previously bitten by fleas was lacking in patients who were able to give a clear history of an

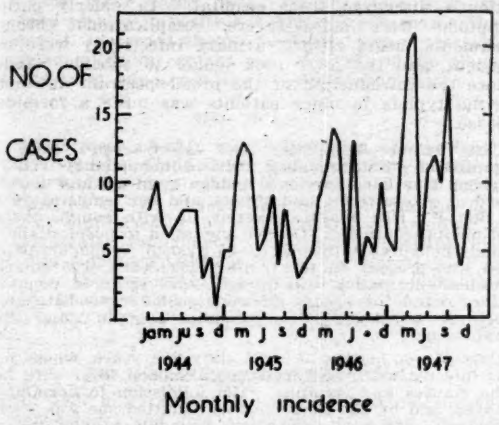

FIGURE III.
Seasonal incidence of murine typhus in Western Australia in the years 1944-1947.

isolated contact with rats. In fact, two patients who regularly produced weals at the site of flea and mosquito bites were adamant that they had not been flea-bitten. On the other hand, there was often fairly convincing evidence that infection had occurred by the respiratory route as a

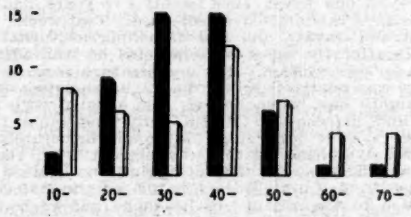

FIGURE IV.
Age and sex incidence among 96 patients with typhus fever admitted to the Royal Perth Hospital, 1944 to 1953. White columns, females; black columns, males.

result of the inhalation of infected dust. Frequently encountered stories were those of waitresses whose job was to sweep the floors of rat-infested cafés, of upholsterers handling kapok, the seeds of which are a favourite source of food to rats, of housewives whose only contact with rats was to sweep and dust the infested rooms, of poultry keepers tearing down old wooden fowl houses, and so on, the common denominator being exposure to dust in rat-infested premises rather than more intimate contact with rats and rat fleas.

In several instances it was possible to assess the incubation period at between nine and fourteen days.

Symptomatology.

The course of a typical infection is shown in Figure V.

The onset of the illness is abrupt, with unabating headache, pains in the back and aching in the limbs, severe malaise, and obstinate constipation. The appetite is greatly impaired and vomiting may occur. A harsh cough often develops in the first week; rigors and drenching sweats finally force the patient to bed. The characteristic exanthem appears after the fifth day; with its fading several days later constitutional symptoms gradually subside. The fever is intermittent and falls by lysis late in the second or in the third week. As in typhoid fever, elevation of temperature is associated with relative bradycardia in uncomplicated cases.

TABLE I.
Occupations and Probable Source of Infection in 82 Cases of Murine Typhus.

Occupation or Source of Infection.	Number of Cases.
Warehouse, shop and café workers (shop assistants, packers, clerks *et cetera*)	26
Occupations related to farming	6
Mixed occupations with casual contact with rats (truck drivers, plumbers *et cetera*)	11
Contact with rats in the home ..	39

A detailed analysis of symptoms and signs in the cases studied is shown in Figure VI.

Headache was usually of frontal distribution, and with the association of some neck stiffness and occasional photophobia often simulated that of meningitis. It was not readily relieved by the common analgesics. Malaise was severe. Gastro-intestinal symptoms during the pre-exanthematous stage were often striking and misleading.

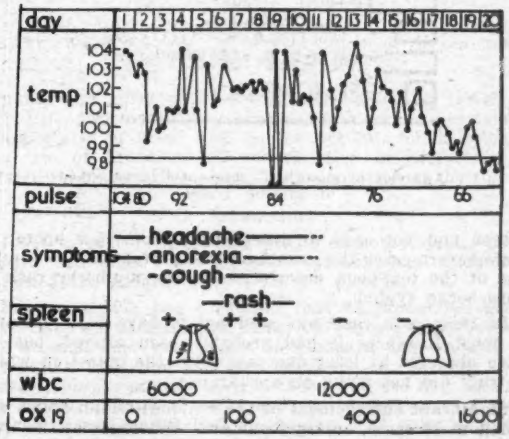

FIGURE V.
Course of infection in a male patient, aged thirty-seven years, admitted to hospital on the second day with a provisional diagnosis of acute frontal sinusitis.

Anorexia, nausea and vomiting presented great difficulties in the nursing of elderly enfeebled patients. Although constipation was the rule, diarrhœa was not uncommon. Upper respiratory tract symptoms were common. Initial sore throat or coryzal complaints and a harsh cough developing at about the time of the appearance of the rash were the usual symptoms. Rarely was the cough productive of sputum; when this occurred there was generally good reason to suspect secondary bacterial infection or the pulmonary congestion of heart failure. Two patients complained of transient pleural pain. Symptoms of severe central nervous system involvement were uncommon. In nine cases irrational behaviour, hallucinations or drowsiness pointed to cerebral abnormality; but the patients were old, with severe dehydration and cerebral arteriosclerosis. Coma is rare.

Physical Signs.

The flushed face and slightly injected conjunctivæ presented a characteristic appearance.

The rash usually appeared five, six or seven days after the onset of symptoms. There were several authentic instances in which the rash made a later appearance, on the tenth, eleventh and twelfth days. Discrete raised papules and macules, fading on pressure, varying in diameter from 1·0 millimetre to 0·5 centimetre, appeared first on the trunk and back, spreading to the proximal parts of the limbs. Sometimes lesions had a purpuric quality. Fresh crops appeared in waves in more severe cases. Fading occurred within forty-eight hours, leaving patches of brown discoloration. In mild cases lesions were

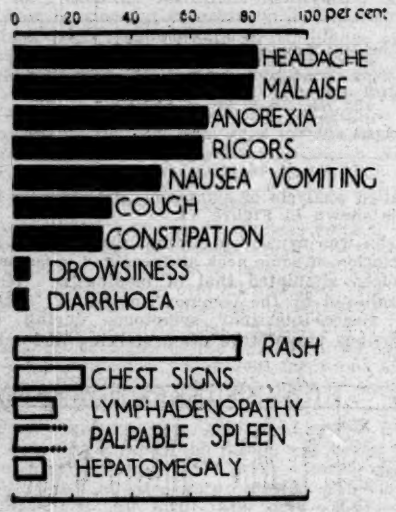

FIGURE VI.

Analysis of signs and symptoms in 96 cases of murine typhus.

sparse and not easy to differentiate from rose spots; in more severe cases the exanthem was profuse and reminded one of the text-book description of the mulberry rash of louse-borne typhus.

In 20 cases a rash was said not to have been present; in most instances it had probably been missed, but we have observed at least one case from the outset in which it truly did not make an appearance.

Significant enlargement of the regional lymph nodes was noted in 12 cases, enlargement and tenderness of the liver were present in 10, and the spleen was palpable in 11. More careful daily inspection of the abdomen might have revealed a higher incidence of splenic and hepatic enlargement.

Approximately one-quarter of the patients had significant signs in the chest—scattered rales throughout both lung fields—attributable to the infection. In five elderly patients signs of consolidation in the lower lobes were attributed to secondary bronchopneumonia or to congestive heart failure, or to a combination of both. X-ray examinations of the chest were not made as a routine procedure; usually films were taken of patients with symptoms and signs referable to the lungs. Of a series of 22 films examined, significant radiological changes, clearing with the recovery of the patient, were present in six. These X-ray signs consisted of a moderate increase in density of the bronchovascular markings in the lung fields (see Figure VII) and, in five cases, diffuse hazy opacities of non-segmental distribution (Figures VIII and IX) in the middle and lower zones referred to as "pneumonitis" by reporting radiologists. These opacities were not unlike the ground-glass radiological appearances seen in other virus and rickettsial types of pneumonia (for example, "Q" fever and ornithosis).

Course of the Disease.

A close parallel existed between the age of the patient and the severity of the infection. In younger patients the disease ran a fairly benign course, without complications, the fever usually subsiding at the end of the second week. However, convalescence was often slow and tedious, and return to work delayed for three or four weeks after the patient's discharge from hospital. In elderly patients symptoms were more severe, complications (bronchopneumonia, heart failure, urinary infections) were more frequent, and the fever took longer to subside. Indeed, before the introduction of the broad-spectrum antibiotics, murine typhus in older patients was quite a formidable disease.

CASE 94.—A housewife, aged sixty-seven years, had organized a "blitz" against rats in her poultry run, and thirteen days later developed sudden vomiting and diarrhœa. She had severe rigors and sweats, and her temperature rose to 104° F. She became severely ill, with cough, dyspnœa and mental confusion. On her admission to hospital she was grossly dehydrated, dyspnœic and flushed. A maculo-papular rash was present on the trunk. The heart was enlarged; auricular fibrillation was present and signs of congestive failure called for urgent attention. She responded immediately to treatment with oxygen, "Digoxin" and chloramphenicol.

CASE 65.—A housewife, aged sixty-five years, whose house was infested with rats, developed a sudden fever with headache, nausea and vomiting. Her admission to hospital was delayed, and by the time she was admitted she was severely dehydrated and semi-comatose. A maculo-papular rash was present on the trunk. Signs of consolidation of the lower lobe of the right lung were present. The urine contained a few pus cells and the blood urea level was 80 milligrammes per 100 millilitres. Despite treatment with penicillin and sulphonamides (broad-spectrum antibiotics were not then available) her condition worsened. She died in a coma (probably uræmic) a week after her admission to hospital. No autopsy was performed.

Occasionally the course of the disease followed an unusual pattern which rendered clinical diagnosis extremely difficult.

CASE 96.—A bus driver, aged twenty-two years, had contact with rats in a rat-infested wheat shed. Two weeks later he felt faint and unwell, and had drenching nocturnal sweats. Two days after the onset of symptoms he was admitted to hospital for observation. His temperature rose to 104° F. and there was relative bradycardia. A harsh cough developed on the eighth day, when he was also afflicted with extreme nausea and diarrhœa. Careful daily inspection failed to reveal a rash at any stage, although moderately severe generalized lymphadenopathy was present from the eighth to the twelfth day. The titre of agglutinins against *Proteus* OX19 rose from 0 initially to 1 in 400 on the sixteenth day. In addition to the absence of headache and rash, the case was unusual in that neither chloramphenicol nor aureomycin influenced the pyrexia, which subsided by lysis at the end of the third week.

Laboratory Findings and Pathology.

The Blood Count.

In most cases a moderate degree of anæmia (hæmoglobin value 9 to 11 grammes *per centum*) was demonstrable during the second week of infection. Slight leucopenia was usually present in the first week and was followed by moderate polymorphonuclear leucocytosis (8000 to 16,000 cells per cubic millimetre) during the second week. The degree of leucocytosis paralleled the severity of infection.

Cerebro-spinal Fluid.

Lumbar puncture was performed on seven patients during the first week owing to the suspicion of meningitis. In no case was an increase of cells or protein content noted.

Liver Function Tests.

Tests of liver function have rarely been called for except in a few cases recently studied. In the rather atypical Case 96 quoted above, tests of liver function performed on

the fourteenth day gave highly abnormal results (serum bilirubin content 0·2 milligramme *per centum*, serum alkaline phosphatase content 13·4 King-Armstrong units, thymol flocculation "++++", thymol turbidity five units, serum albumin content 2·5 grammes *per centum*, serum globulin content 4·1 grammes *per centum*). Evidence of liver damage was confirmed by liver biopsy, which revealed focal areas of necrosis of liver cells in the mid-zonal regions of the lobule, with an acute inflammatory reaction comprising polymorphonuclear cells, eosinophile cells, plasma cells and macrophages (Figure X).

In four other cases liver function tests have given abnormal results: a positive response to the thymol flocculation test, increased thymol turbidity, and depression of the serum albumin content with reversal of the albumin-globulin ratio have been the consistent pattern.

Skin Biopsy.

A small piece of skin was taken from one patient for examination of sections at the first appearance of the rash, before a positive titre against *Proteus OX*19 was detected. Proliferation of the endothelium with obstruction of the lumen of capillaries in the corium and a surrounding zone of perivascular inflammatory reaction, comprising histiocytes and polymorphonuclear leucocytes, was seen (Figure XI). In the hæmatoxylin-stained sections ovoid bodies 1μ to 2μ in size were seen in the cytoplasm of many endothelial cells. These were, in all probability, rickettsial bodies.

The Weil-Felix Agglutination Test.

From 1927 onwards the *Proteus OX*19 strain in use in the laboratory was one originally obtained in Kuala Lumpur. By the late war this strain had gone irretrievably "rough", and a fresh strain obtained from the Commonwealth Serum Laboratory has been subsequently used. Antigen is prepared by conventional alcohol treatment of agar cultures, and saline suspensions ready for use are preserved with chloroform. Prior to 1949 routine tests covered a range of dilution of 1 in 100 to 1 in 800; since then lower dilutions (1 in 25 and 1 in 50) have been added. Test material is incubated for two hours at 55° C. and overnight at 37° C. Zones of inhibition have not been observed.

The Weil-Felix test is performed as a routine measure, whether or not typhus fever is suspected clinically, on all sera submitted to this laboratory for agglutination tests in the diagnosis of obscure fever. Since the lower dilutions of antigen have been included in routine testing, a number of low-titre agglutinations (1 in 25 and 1 in 50) have been detected in patients not suffering from clinical typhus fever—48 out of 264 sera tested in 1952, and 71 out of 270 in 1953. The brief clinical notes did not indicate whether or not past infection with typhus (either louse-borne or murine) had occurred, whether patients had received anti-typhus inoculations, or whether incidental *Proteus vulgaris* urinary tract infection was present, so that this finding—that positive results to agglutination tests in low titres are present in 20% of the population—is not readily explained. That such low titres are found in persons who have previously had louse-borne and murine typhus is known; we have records of two European migrants who contracted epidemic typhus in 1919 and 1941, with residual titres of 1 in 25 and 1 in 50 respectively.

The reliability of the Weil-Felix test in the diagnosis of typhus has been reviewed by Felix (1944), who regarded a rise in titre from 0 to even as low a dilution as 1 in 100 as of significance. The results of agglutination tests in cases of murine typhus are shown in Figure XII.

Agglutinins are usually detectable seven to ten days after the onset of fever, reaching a maximum titre of 1 in 800 or 1 in 1600 about the end of the second week. Titres begin to fall in the fourth or fifth week, and the sera of most patients examined six months after infection have been found to have titres not higher than 1 in 25 or 1 in 50. Occasionally high titres persist; one patient has maintained a titre of 1 in 800 six months after infection.

Slight confusion in the serological diagnosis of murine typhus occasionally arose as a result of anamnestic rise in titres of agglutinins against typhoid and paratyphoid H and O antigens in patients who had previously received inoculations against typhoid and paratyphoid A and B (to titres of 1 in 800), and less frequently of agglutinins against *Brucella abortus* (to 1 in 200).

Differential Diagnosis.

Despite general familiarity with the disease, a correct diagnosis was made before the results of the Weil-Felix reaction were to hand in only 40% of cases. Provisional diagnoses included the following: pyrexia of unknown origin, sinusitis, influenza, tonsillitis, bronchitis, pneumonia, measles and rubella, sulphonamide hypersensitivity,

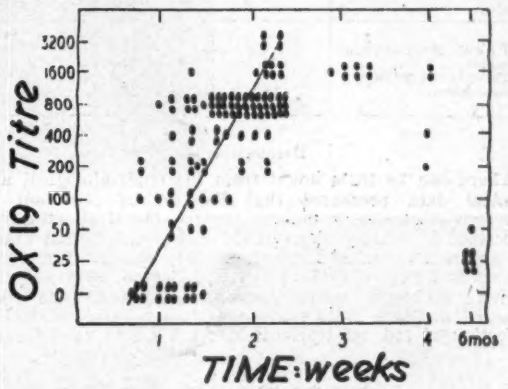

FIGURE XII.

The Weil-Felix reaction: serial titres of agglutinins against *Proteus OX19 in 96 cases.*

typhoid and paratyphoid fever, gastro-enteritis, infectious hepatitis, poliomyelitis, meningitis. The correct provisional diagnosis is of great help to the patient, for it prompts the use of broad-spectrum antibiotics in preference to penicillin, materially shortening the duration of the illness.

Treatment.

Prior to 1949 reliance was placed on symptomatic treatment; but since then specific therapy has completely modified the natural history of the disease.

Snyder (1948) has indicated that sulphonamide drugs may have an unfavourable effect on the course of typhus. An attempt has been made to compare the severity of symptoms and the duration of fever in patients who received sulphonamides and in those who did not. Results are shown in Table II. The apparent significance of these figures is perhaps vitiated by the fact that sulphonamides were used in the treatment of many severely ill patients, and less frequently in mild cases; nevertheless, there is enough clinical evidence to suggest that sulphonamides are not only useless in rickettsial infections, but may be actually harmful.

The use of para-aminobenzoic acid (PABA) in typhus fever was reported on by Yeomans, Snyder, Murray and Zarafonetis in 1944, and is now of historical interest only. Four patients were treated with PABA in 1949 and 1950. In only one case was any significant reduction of fever noted.

Since 1950, in view of the reports of Smadel and Jackson (1947) and of Snyder *et alii* (1950), the broad-spectrum antibiotics have been used—chloramphenicol in seven cases, aureomycin in three, and "Terramycin" in one. Each antibiotic has been equally effective, causing a fall in temperature in forty-eight hours when a loading dose of three grammes was given and followed by 500 milligrammes every six hours. In two cases treatment was begun at the time of the appearance of the rash and was continued for

only four days; a relapse, with recurrence of fever, symptoms and rash, recurred within four days of the interruption of treatment (Figure XIII). When treatment is begun before antibodies have reached their peak, it should be continued for at least four or five days after the fall in temperature. In only one case (Case 96) has fever not subsided on treatment with conventional dosages of broad-spectrum antibiotics.

TABLE II.

A Comparison of the Course of Infection in Patients with Murine Typhus Treated with Sulphonamides and Those who did not Receive Sulphonamides.

Group.	Mean Duration of Fever. (Days.)	Range. (Days.)	Number of Patients with Severe Complications.
50 Patients given sulphonamides	23	18 to 32	5
36 Patients not given sulphonamides	18	11 to 23	0

Discussion.

There can be little doubt from the epidemiological and clinical data presented that the typhus prevalent in Western Australia is murine typhus, identical with that described in other regions of the globe—the United States of America (Lee, 1912; Paullin, 1913; Maxcy, 1926, 1929; Stuart and Pullen, 1945; Miller and Beeson, 1946), Mexico (where *Rickettsia mooseri* was first isolated), and other Australian States. Murine typhus is a natural subclinical infection of rodents transmitted from rat to rat by their

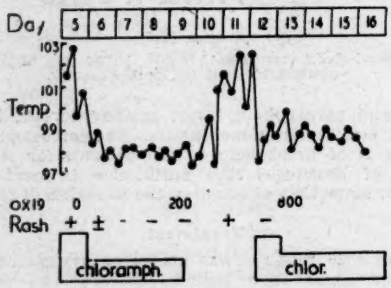

FIGURE XIII.

Relapse as a result of inadequate antibiotic treatment.

ectoparasites, the rat louse (*Polyplax spinulosus*) or the flea (*Xenopsylla cheopsis*). It is presumed that a high proportion of rats in many areas of Western Australia are parasitized with Rickettsiæ, and evidence has been adduced that the incidence of infection in man, which is an incidental occurrence, follows geographically the predicted aggregation of rodent population near the warehouses and granaries of the main ports of the metropolitan area and in country towns. With regard to the mode of transmission from rat to man, it has been shown in the present study that there is a considerable body of evidence to suggest that infection via the respiratory route is of clinical importance. Rickettsial particles remain viable in the dry state for a considerable time, and it is known that infections in laboratory workers — among whom rickettsioses have acquired an unenviable reputation — are acquired by the respiratory route. In a high proportion of the cases we have encountered the illness had apparently followed exposure to infected dust.

The incidence of murine typhus is indeed higher in Western Australia than in any other State of the Commonwealth. In South Australia annual notification since 1932 has never exceeded 14 cases, with a mean of seven cases a year. In Queensland the peak incidence of all forms of typhus (including scrub and tick-borne typhus) occurred in 1944-1945 (Annual Report, Commissioner of Public Health, 1945), when 104 cases were recorded. In New South Wales notifications of typhus fever in the years 1942, 1943, 1944 and 1945 were 8, 16, 30 and 26 respectively (Report of the Director-General of Public Health, 1941-46, 1953).

Murine typhus is commonly regarded as a mild disease, in contrast to scrub and louse-borne typhus. However, an age-specific mortality rate of nearly 10% in persons aged over fifty years cannot be regarded lightly by physicians, who cannot fail to be impressed by the duration of fever and high incidence of complications in this age group. Now that it is possible to modify the course of infection abruptly with broad spectrum antibiotics, the diagnosis of typhus from other fevers before the Weil-Felix reaction is positive becomes a matter of importance. The abrupt onset with headache and grippal pains, followed by anorexia, nausea, obstinate constipation and/or upper respiratory tract symptoms in a person who has been in contact with rats, should suggest the diagnosis of typhus even before the characteristic rash appears, and should call for the administration of aureomycin, "Terramycin" or chloramphenicol until the diagnosis is substantiated on serological grounds. The use of penicillin in the interim is useless and there is reason to suppose that sulphonamides may be actively harmful.

There is no evidence that any one broad-spectrum antibiotic is preferable to others. Provided that simple rules of dosage are observed, prompt recovery almost invariably occurs. A substantial initial loading dose (three grammes) should be given, and treatment should be continued (500 milligrammes every six hours) for at least five days after the fall in temperature.

It has not been possible to make any fundamental contribution to the understanding of the pathology of murine typhus, about which there exists a paucity of information. It is probably similar — only in lesser degree — to that occurring in epidemic typhus (see Karsner, 1953), in which it is known that multiplication of Rickettsiæ occurs in the endothelial cells of small blood vessels, causing thrombosis and focal ischæmia in the skin, kidneys, myocardium, brain and other organs. The accumulations of polymorphonuclear cells and macrophages in small ischæmic areas have been referred to as Fraenkel's nodes. The finding of such areas of necrosis in a liver biopsy specimen, and of characteristic lesions in the skin, and radiological evidence of interstitial pneumonic changes, points to the probability of similar widespread changes in the vascular system being present in murine typhus.

The relevant literature has been quoted to indicate that Brill's disease is not murine typhus. For the sake of accuracy the former eponym could well be dropped from popular local usage. However, the possibility of recrudescent typhus appearing among the large number of migrants entering Australia, some of whom give a history of having previously been infected with *R. prowazeki*, must be considered. Apart from New York (Murray *et alii*, 1950) true Brill's disease has been described in England (Hawksley and Stokes, 1950) and in Europe (Girond, Boyer and Varques, 1950) since 1945. Clinically there is little to distinguish Brill's disease from murine typhus, except that a rash is often absent and fever is of shorter duration. However, in Brill's disease the Weil-Felix test produces a negative result, and diagnosis is based on the detection of a rise in titre of complement-fixing antibodies against *R. prowazeki*.

Summary.

1. The incidence of endemic typhus is higher in Western Australia than in any other State of the Commonwealth. Since 1927, 1332 cases have been reported, with 50 deaths. From 1945 to 1948 annual notifications in the State exceeded 100 cases.

2. Geographically the chief foci of infection are Perth and Fremantle; but the disease is widespread in the State, occurring as far north as Geraldton and as far south as Albany.

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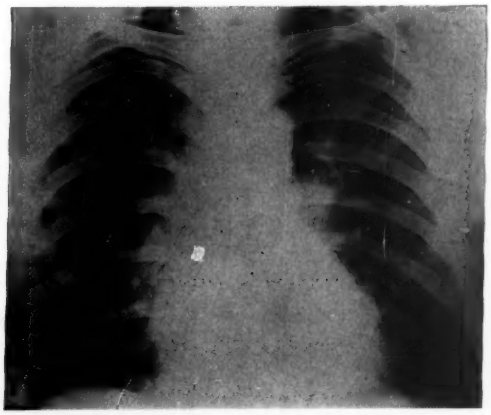

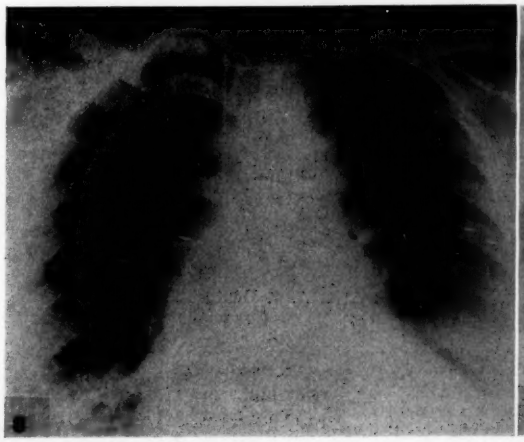

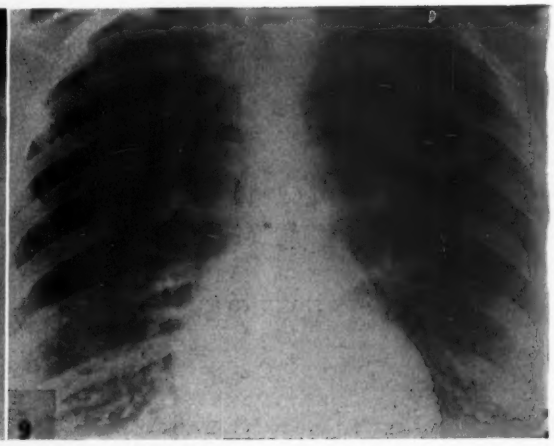

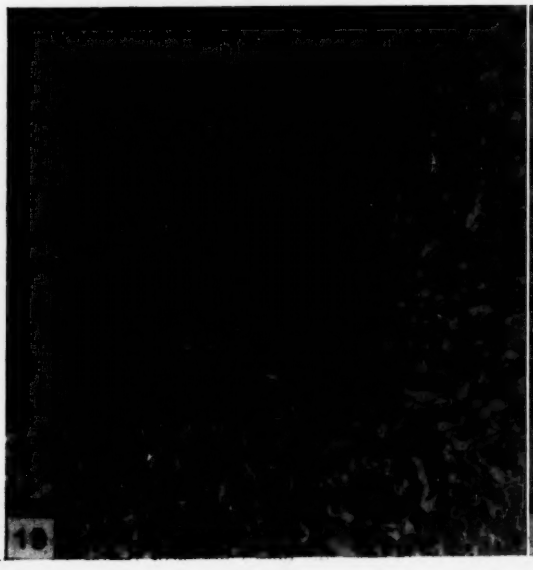

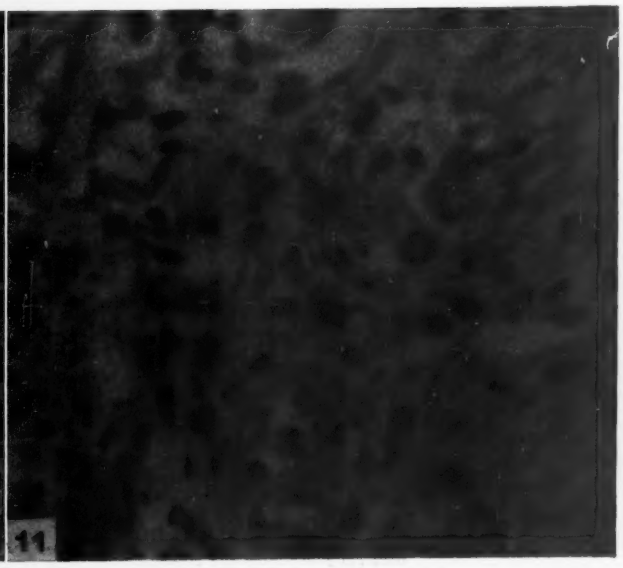

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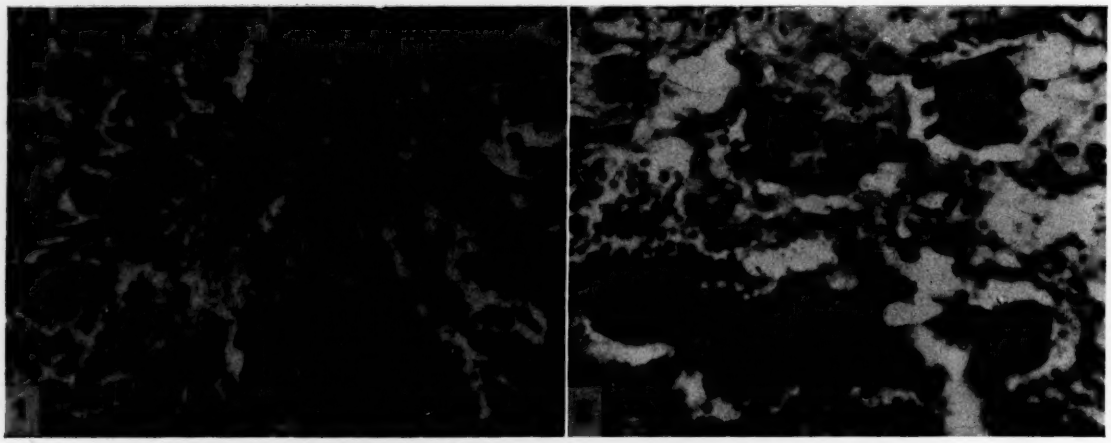

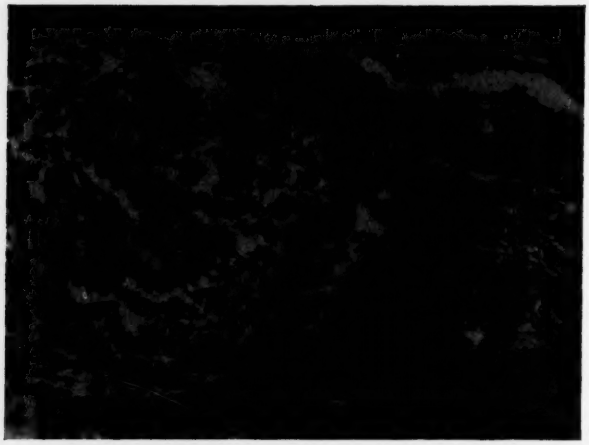

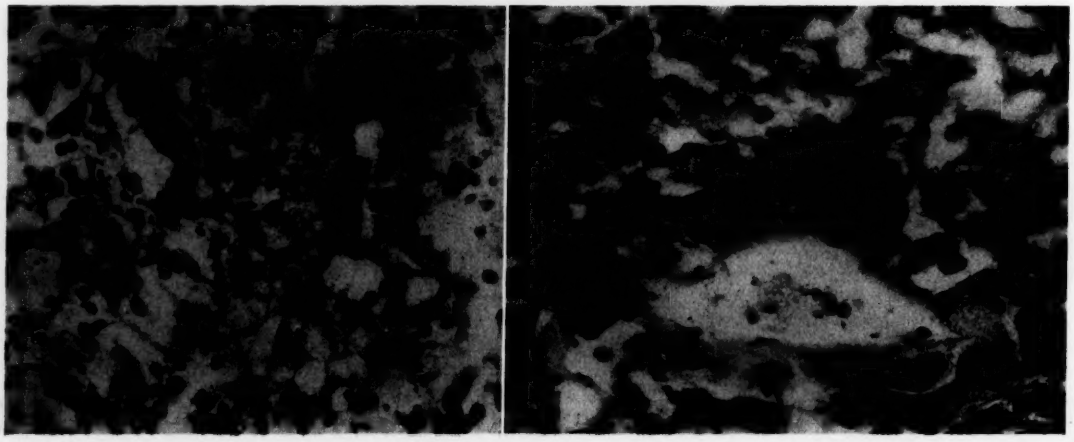

3. There is a seasonal preponderance of cases during the summer and autumn months.

4. The almost universal history of contact with rats identifies the fever as murine typhus. Moreover, the clinical features are in close conformity with other classical descriptions of murine typhus. There is suggestive evidence that in many cases infection was acquired by the respiratory route.

5. Although a relatively mild infection in young people, endemic typhus has been the cause of considerable morbidity and not inconsiderable mortality in elderly patients.

6. The disease is of abrupt onset with grippal symptoms, and often with respiratory and gastro-intestinal manifestations. A few patients have radiological signs of rickettsial pneumonitis. The maculo-papular rash usually appears on the fifth to ninth day. Fever subsides by lysis in the third week. Titres of between 1 in 400 and 1 in 1600 of agglutinins against *Proteus OX19* develop late in the second week.

7. There is evidence that the use of sulphonamides is contraindicated in typhus fever.

8. Aureomycin (chlortetracycline), chloramphenicol and "Terramycin" (oxytetracycline) are all of equal efficacy in causing the abrupt termination of fever, symptoms and signs.

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References.

ANDREW, R., BONNIN, M., and WILLIAMS, S. (1946), "Tick Typhus in North Queensland", M. J. AUSTRALIA, 2: 253.

ANNUAL REPORT (1945), Commissioner of Public Health, Queensland.

BRILL, N. E. (1910), "An Acute Infectious Disease of Unknown Origin. A Clinical Study Based on 221 Cases", *Am. J. M. Sc.*, 139: 484.

BURNET, F. M. (1942), "The Rickettsial Diseases in Australia", M. J. AUSTRALIA, 2: 129.

BURNET, F. M., and FREEMAN, M. (1937), "Experimental Studies on the Virus of 'Q' Fever", M. J. AUSTRALIA, 2: 299.

CUMPSTON, J. H. L., and MCCALLUM, F. (1927), "The History of Intestinal Infections (and Typhus Fever) in Australia, 1788-1923", Commonwealth of Australia, Department of Health Service Publication, Number 36.

DERRICK, E. H. (1937), "'Q' Fever: A New Fever Entity: Clinical Features, Diagnosis and Laboratory Investigation", M. J. AUSTRALIA, 2: 281.

DRUMMOND, A. F. (1953), unpublished observations.

DYER, R. E., RUMREICH, A., and BADGER, L. F. (1931), "Typhus Fever. A Virus of the Typhus Type Derived from Fleas Collected from Wild Rats", *Pub. Health Rep.*, 46: 334.

FELIX, A. (1944), "Technique and Interpretation of the Weil-Felix Test in Typhus Fever", *Tr. Roy. Soc. Trop. Med. & Hyg.*, 37: 321.

FUNDER, J. F., and JACKSON, A. V. (1946), "North Queensland Tick Typhus: A Comparative Study of the Rickettsia with that of Murine Typhus", M. J. AUSTRALIA, 2: 258.

GIROND, P., BOYER, J., and VARQUES, R. (1950), "*A propos de quelques cas de typhus exanthématique survenus chez des sujets non parasités*", *Presse méd.*, 58: 334.

HAWKSLEY, J. C., and STOKES, E. J. (1950), "A Case of Brill's Disease in London", *Lancet*, 2: 97.

HEASLIP, W. G. (1940), "An Investigation of the Condition Known as Coastal Fever in North Queensland and its Separation from Scrub Typhus", M. J. AUSTRALIA, 2: 555.

HEASLIP, W. G. (1941), "Tsutsugamushi Fever in North Queensland, Australia", M. J. AUSTRALIA, 1: 380.

HONE, F. S. (1922), "A Series of Cases Closely Resembling Typhus Fever", M. J. AUSTRALIA, 1: 1.

HONE, F. S. (1923), "A Further Study of Cases Closely Resembling Typhus Fever", M. J. AUSTRALIA, 1: 435.

KARSNER, H. T., and others (1953), "Pathology of Epidemic Typhus: Report of Fatal Cases Studied by United States of America Typhus Commission in Cairo, Egypt, during 1943-45", *Arch. Path.*, 56: 397.

LANGAN, A. M., and MATHEW, R. Y. (1935), "The Establishment of 'Mossman', 'Coastal' and Other Previously Unclassified Fevers of North Queensland as Endemic Typhus", M. J. AUSTRALIA, 2: 145.

LEE, R. I. (1913), "Typhus Fever (Brill's Disease) at the Massachusetts General Hospital in Ten Years (Oct. 1, 1902, to Oct. 1, 1912)", *Boston M. & S. J.*, 168: 122.

MATHEW, R. Y. (1938), "Endemic Typhus in North Queensland", M. J. AUSTRALIA, 2: 371.

MAXCY, K. F. (1926), "Clinical Observations on Endemic Typhus (Brill's Disease) in the United States", *Pub. Health Rep.*, 41: 1213.

MAXCY, K. F. (1929), "Typhus Fever in the United States", *Pub. Health Rep.*, 44: 1735.

MILLER, E. S., and BEESON, P. B. (1946), "Murine Typhus Fever", *Medicine*, 25: 1.

MOOSER, H., CASTANEDA, M. R., and ZINSSER, H. (1931), "Rats as Carriers of Mexican Typhus Fever", *J.A.M.A.*, 92: 231.

MURRAY, E. S., BABHR, G., SCHWARTZMAN, G., MANDELBAUM, R. A., ROSENTHAL, N., DOONE, J. C., WEISS, L. B., COHEN, S., and SNYDER, J. C. (1950), "Brill's Disease. I. Clinical and Laboratory Diagnosis", *J.A.M.A.*, 142: 1059.

PAULLIN, J. E. (1913), "Typhus Fever, with a Report of Cases", *Southern M. J.*, 6: 36.

PLOTZ, H. (1943), "Complement Fixation Test in Rickettsial Diseases", *Science*, 97: 20.

PLOTZ, H., SMADEL, J. E., BENNETT, B. L., REAGAN, R. H., and SNYDER, M. J. (1946), "North Queensland Tick Typhus: Studies of the Ætiological Agent and its Relation to Other Rickettsial Diseases", M. J. AUSTRALIA, 2: 263.

SMADEL, J. E., and JACKSON, E. B. (1947), "Chloromycetin, an Antibiotic with Chemotherapeutic Activity in Experimental and Rickettsial Infections", *Science*, 106: 418.

SNYDER, J. C. (1948), "The Treatment of the Rickettsial Diseases in Man", Symposium on Rickettsial Diseases, American Association for the Advancement of Science, Boston, 1946.

STUART, B. M., and PULLEN, R. L. (1945), "Endemic (Murine) Typhus Fever: Clinical Observations of 180 Cases", *Ann. Int. Med.*, 23: 520.

WHEATLAND, F. T. (1926), "A Fever Resembling a Mild Form of Typhus Fever", M. J. AUSTRALIA, 1: 261.

YEOMANS, A., SNYDER, J. C., MURRAY, E. S., ZARAFONETIS, C. J. D., and ECKE, R. S. (1944), "The Therapeutic Effect of Para-aminobenzoic Acid in Louse-borne Typhus Fever", *J.A.M.A.*, 126: 349.

ZINSSER, H. (1934), "Varieties of Typhus Virus and the Epidemiology of the American Form of European Typhus Fever (Brill's Disease)", *Am. J. Hyg.*, 20: 513.

Legends to Illustrations.

FIGURE VII.—X-ray film of the chest of a patient with murine typhus, showing prominent increase of bronchovascular markings.

FIGURE VIII.—X-ray film of the chest of a patient with murine typhus, showing hazy opaque area in the left lower zone: rickettsial pneumonitis.

FIGURE IX.—Opaque areas of "ground glass" appearance in the lower zones of both lungs in a patient with murine typhus fever.

FIGURE X.—Liver biopsy of a patient with probable murine typhus, showing zone of recent necrosis of polygonal cells and invasion of area by polymorphonuclear cells, eosinophile cells, plasma cells and macrophages.

FIGURE XI.—Section taken through a small papule on the forearm of a patient with murine typhus showing a small "granulomatous" lesion in the corium. The capillary endothelium is swollen, and there is a focal accumulation of macrophages and polymorphonuclear leucocytes.

FISTULOUS IMPERFORATE ANUS.

By PETER ILBERY,

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IMPERFORATE ANUS and the fistulæ associated with it are anomalies occurring between the fifth and tenth weeks of intrauterine life.

Embryology.

In an embryo of 3·5 millimetres the cloaca appears as a sort of conical sac receiving the wider-lumened hind gut dorsally, and the narrow-lumened allantois ventrally. Ventrally it is limited for about half its extent by the epithelial cloacal membrane. The walls of the cloaca present a well-marked groove, representing the later line of division, extending from the saddle between the allantois and the hind gut to about the middle area of the cloacal membrane. The part of the cloaca caudad to the cloacal membrane—the tail gut—is short and cone-shaped. In the 5·0 millimetre embryo considerable degeneration

of the tail gut has occurred. The anomaly where the rectum ends blindly above the perineum is attributed by Boyden to excessive degeneration of this structure. It is thought rather that imperforate anus occurs as a result of a fistula formation; the gut, having once found an aberrant opening to the exterior as outlined below, has no stimulus to open at the proctodæum. (Fistulæ were proven to be present in only 35% of the cases presented; they may well have closed prior to birth or may remain as fibrous connexions.) Such an idea would be in keeping with Sir Arthur Keith's theory of reversion to a more primitive cloaca, such as that in the amphibians.

In a 12·0 millimetre embryo the cloaca is about half divided along the groove. If closure is not later complete, a fistula remains connecting the rectum to the bladder or urethra.

In the female the two Müllerian ducts develop and form the uterus and vagina. As the vagina finds its way to the perineum, it takes over a preexisting recto-urinary communication resulting in a recto-vaginal fistula.

In the 16·0 millimetre embryo the cloaca and cloacal membane are completely divided, the ventral segment being limited by the uro-genital plate, and the dorsal segment by the anal plate. The great increase in pre-cloacal tissue has not only resulted in the genital eminence, but the uro-genital plate has been dislocated into the base of the phallus. It is thought that in the case of the recto-perineal and recto-*fossa navicularis* fistulæ, the terminal part of the gut is thus displaced anteriorly, and in those cases such as the one recorded it was further displaced anteriorly by the developmental process which causes the prolongation of the cloaca to form the urethra.

Fortunately for the repair of these anomalies, the end sphincter is normally developed, in all cases, as it arises in connexion with the perineum and not with the bowel.

It would then appear embryologically (as well as on the grounds of prognosis and treatment) that the associated fistulæ can be divided into the following three groups: the recto-vesical and recto-urethral; the recto-vaginal; and the recto-*fossa navicularis* and recto-perineal.

Classification.

In a series of 40 cases of congenital abnormalities of the anus and rectum at the Royal Alexandra Hospital for Children over the last ten years, males have been predominantly affected (three to one female). When the classification of Ladd and Gross is used, there have been two cases of Type I (one in a female with a recto-perineal fistula to the right side of the mid-line), two of Type II, and 35 of Type III. No case of Type IV was recorded, and there was one case of covered anus. There were 13 cases of fistulæ, 12 of the 35 patients classified in Type III having this associated finding. The recto-urethral fistula in the female occurred with an imperforate vagina. Frank fistulous imperforate anus was twice as common in the female as in the male.

Radiological Considerations.

Wangensteen and Rice first pointed out the value to the surgeon, faced with a tedious and seemingly inaccessible approach from beneath, of corroborative evidence in the form of a radiograph taken with the patient in the inverted position of the distance of the uppermost gas bubble from an anal marker. There are three points of importance in this examination. The first is that the anal marker should be firmly in contact with the anal dimple. The second is that the infant should be inverted for some minutes before being radiologically examined. (I have recently seen the gas bubble approach from over three-quarters of an inch in the first radiogram to a point half an inch from the proctodæum in the second radiogram after inversion of the baby for five minutes.) The third is that the procedure is not feasible until twelve hours after birth, when gas will have found its way into the large intestine. Associated deformities are very common, and in the region radiologically examined at this time evidence of anomalies of the sacrum and hemivertebræ should be sought.

Report of a Case.

Baby X., a male, was admitted to hospital on June 6, 1954, having been born the previous evening at 8 p.m. The pregnancy had been normal, but the mother when approximately twenty-eight weeks pregnant had been admitted to hospital because of a mild ante-partum hæmorrhage. She subsequently came into labour spontaneously at full term on June 5, and labour was normal.

TABLE I.

Type of Fistula.	Males.	Females.	Total.
Class I:			
Recto-perineal	3	1	5
Recto-*fossa navicularis*	—	1	
Class II:			
Recto-vesical	3	—	6
Recto-urethral	2	1	
Class III:			
Recto-vaginal	—	2	2
Total	8	5	13

On examination, the baby had an imperforate anus. It was at first thought that there was no urethral orifice, only a slight dimple being present at the normal site. There was a linear thinning of the mid-line raphe of the scrotum a quarter of an inch posterior to the base of the penis. On palpation of the scrotum crepitus could be elicited. The provisional diagnosis was imperforate anus and urethra.

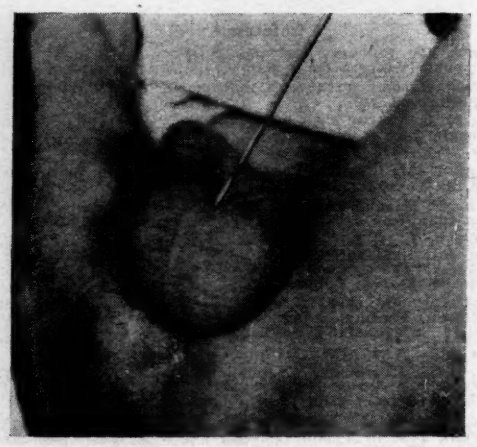

FIGURE I.
Photograph with probe directed into recto-perineal fistula.

A radiographic examination was made at midday, and revealed the following abnormalities. An imperforate anus was present. The gas bubble in the most distal part of the large intestine was half an inch from the marker at the proctodæum. There appeared to be a thin column of air outlining the urethra, which it was thought might end in the scrotum. The appearances suggested a proximal communication with the large intestine, but this could not be demonstrated in the plain X-ray film.

That evening a small incision was made in the thin wall of the sac protruding in the mid-line a quarter of an inch behind the penis. Air escaped through the hole, and at the end of the procedure urine was passed through the terminal penile dimple.

The following day meconium was passed through the fistula. At operation on June 8 a probe was passed back along the fistula into the rectum and then directed to the anal dimple, and an incision was made about it. The rectal mucosa was then stitched to the anal skin by interrupted sutures. X-ray examination after the injection of "Lipiodol" into the fistula outlined it from the scrotum as it pursued its way into the lowermost part of the rectum. It was super-

ficial in its early course, but finally lay at its junction with the rectum, about half an inch from the proctodæum, which was the level of the lowermost gas bubble seen in the previous radiograph. The X-ray appearance was that of imperforate anus with recto-perineal fistula into the scrotum.

The interest in this case lies in that it is extremely rare to have such an anterior opening for a recto-perineal fistula, that it occurred through the scrotum (Ladd and Gross in a large series and other authors have no such case), and that it was covered by a membrane.

Summary.

1. With the exclusion of the stenotic cases of imperforate anus and perhaps of the membranous forms, it is suggested that all those cases of Type III, or those in which the terminal part of the gut is separated by an appreciable distance from the proctodæum, are really cases of fistulous imperforate anus in which the fistula has either closed prior to birth or remains as a fibrous cord or as a frank fistula. The last-mentioned are then classified into three groups on embryological grounds.

2. Thirty-five cases of Type III imperforate anus are reviewed; fistula was evident in 12 of them.

3. The radiological aspect is discussed.

4. The case of a hitherto unreported type of recto-perineal fistula is presented.

Acknowledgement.

I wish to thank Dr. E. Goulston for his help and advice, and for permission to discuss this case, the patient having been admitted to hospital under his care.

References.

CROWELL, E. A., and DULIN, J. W. (1940), "Congenital Anomalies of the Anus and Rectum", *Surgery*, 7: 529.

DMYTRYK, E. T. (1945), "Congenital Malformations of the Anus and Rectum", *Arch. Surg.*, 50: 253.

KEITH, A. (1908), "Malformations of the Hind End of the Body", *Brit. M. J.*, 2: 1756.

KEITH, A. (1932), "Malformations of the Human Body from a New Point of View", *Brit. M. J.*, 1: 489.

LADD, W. E., and GROSS, R. E. (1934), "Congenital Malformations of Anus and Rectum', *Am. J. Surg.*, 23: 167.

POHLMAN, A. G. (1911), "The Development of the Cloaca in Human Embryos", *Am. J. Anat.*, 12: 1.

WANGENSTEEN, O. H., and RICE, C. O. (1930), "Imperforate Anus", *Ann. Surg.*, 92: 77.

WOOD JONES, F. (1904), "The Nature of the Malformations of the Rectum and Urogenital Passages", *Brit. M. J.*, 2: 1630.

Reports of Cases.

THORACIC OUTLET SYNDROME; CASE ASSOCIATED WITH SHORT "FIRST" RIB, ANEURYSM OF THE SUBCLAVIAN ARTERY AND OCCLUSION OF BRACHIAL ARTERY.

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ANOMALIES of the thoracic outlet have excited the curiosity of anatomists, physicians and surgeons from early times, cervical rib being described by Galen and Vesalius. The most widely recognized structural abnormalities in this region causing symptoms are accessory (cervical) ribs or unusual positions of first thoracic ribs. The relative importance of the various structures in producing symptoms is not always clear, and it has been suggested that in many patients the *scalenus anterior* muscle plays a prominent part, and overaction of this muscle may be the primary fault; this has led to the use of the term "*scalenus anticus* syndrome". There has been considerable discussion of the mechanism of the vascular symptoms associated with these abnormalities; they have been attributed to disturbance of blood vessels of the arm due to pressure on nerves, to constriction of the subclavian artery, and to damage to the arterial wall resulting in thrombosis with embolism.

Although many examples of vascular disturbance due to abnormalities of the thoracic outlet have been reported, each differs in some respects from the others. The instance reported here is of particular interest—because of clearly defined structural and functional changes and dramatic clinical features.

Clinical Record.

An engineer, aged twenty-seven years, stated that eight weeks prior to his admission to hospital (February, 1953) he began to suffer from attacks of "pins and needles" and pallor of his left ring finger. At first these occurred only over the distal part of the digit, but during the next week the whole digit was affected, and later the adjacent two

FIGURE I.

Photograph of left side of neck. Two pins have been fixed over the aneurysm by cellulose tape and a double exposure taken to show the expansile pulsation.

fingers were affected. Some three weeks after the onset, a similar episode occurred in the left thumb and the left index and middle fingers; after this the attacks were more frequent (occurring four or five times per day) and involved sometimes one group of fingers, sometimes another. They were usually short-lived, and relieved by swinging his arm. However, he had one attack of pallor of his hand lasting twenty minutes, which was associated with faintness. He found that activity of his left hand would induce an attack, and that on prolonged use the hand would become painful and very weak. Holding his hand above shoulder level would cause it to become blanched and cold; when the arm was allowed to hang down, the colour would return.

About a fortnight prior to his admission to hospital he developed tenderness at the base of his left thumb (in the "snuff-box" region) and over the wrist, in the line of the radial artery. He noted that his radial pulse, which had previously been quite strong, had disappeared. From this time he had a dull, aching pain in his forearm on exercising his hand; and after the attacks of pallor he had dull pain beneath his biceps muscle "as the circulation returned". A week after the development of pain in the

wrist region, he found that he could not extend his forearm fully because of a dragging pain in front of his elbow. At first there was no association between the attacks of pallor and exposure to cold, but on the day of his admission to hospital considerable pallor of the hand occurred with the arm in a cold draught.

Apart from the complaints described, the patient was in good health. He had smoked 20 to 30 cigarettes per day for many years, but had stopped three weeks earlier. Except for an attack of mumps seven years previously, he had suffered from no other illnesses or injuries.

On examination, the patient was a generally healthy young man with no physical abnormalities apart from those in his neck and left arm. His blood pressure (in his right arm) was 125 millimetres of mercury, systolic, and 85 millimetres, diastolic. A smooth, pulsatile swelling was visible in the left supraclavicular region (Figure I). The blood pressure was unobtainable in the left arm and forearm. An arterial pulse could be felt to the middle of the arm, but not in the lower part of the brachial artery or the radial vessels, which were felt as tender, cord-like structures. The left forearm and hand were much cooler than the right.

There was no muscle wasting and no abnormal neurological features in his left arm. An attack of blanching was produced by the patient raising his hand above shoulder level. He noted "tingling" in his fingers and a slight ache deep to the biceps muscle. The limb became blanched to the elbow and colder. The patient then said that his hand felt weak, and there was an aching pain in the wrist and palm. When the hand was lowered, the fingers gradually became cyanotic, then a mottled pink and purple and finally bright red, except for the tips which remained cyanosed.

Radiographic examination (Dr. B. L. Deans) revealed 13 ribs on each side. The first of these on the left side was directed more laterally than on the right. It was unusually short and ended proximal to the expected point of crossing of the clavicle and first rib. The anterior end of the second rib on each side was directed towards the normal point of insertion of the costal cartilage of the first rib to the sternum (Figure II). There were seven vertebræ above the highest one bearing a rib, with fusion of the third and fourth vertebral bodies (Figure III), 13 vertebræ bearing ribs, and five lumbar vertebræ. It would seem a fine point in terminology whether the "first" (highest) rib should be labelled a cervical rib (with eight "cervical" vertebræ) or an abnormal first thoracic rib (with 13 "thoracic" vertebræ.)

Course and Further Investigation.

It was considered that the ischæmia was sufficiently gross to require urgent relief, and further investigation was postponed. The following day, under general anæsthesia, a left upper dorsal sympathectomy was performed (Dr. K. Jamieson). There was some improvement in the circulation of his left hand. In view of the probable embolic occlusion of the arteries of the arm, the patient was treated with heparin. Five days after operation, he complained of severe pain in his scapular region and of numbness and blueness of his left hand, and was found to have a large hæmatoma of his back muscles and very severe ischæmia of his hand. Heparin therapy was stopped, the hæmatoma was evacuated and a blood transfusion given. After this, there was a considerable improvement in the circulation of the hand. Further convalescence was uneventful, and the patient was discharged home. Anticoagulant therapy with ethylidene dicoumarol (E.D.C.) was given for several weeks. The circulation in the left hand improved further but did not return to normal; the pulsation in his brachial artery extended a little more distally.

The patient was examined again two months later (April, 1953). No trophic changes had occurred in the hand. Pallor was readily produced when it was elevated and clenched a few times.

Grip Test.—Repetitively squeezing a length of hose attached to a sphygmomanometer to produce a pressure of 200 millimetres of mercury every five seconds produced pain in his thenar and hypothenar eminences after 40 grips (average of six runs).

"Costoclavicular Test".—Traction on the slightly abducted and extended arm on the right side produced obliteration of the radial pulse and on the left side of the subclavian pulse.

"Scalenus Anticus Test".—Adson's manœuvres[1] caused weakening of the right radial pulse and of the left subclavian pulse.

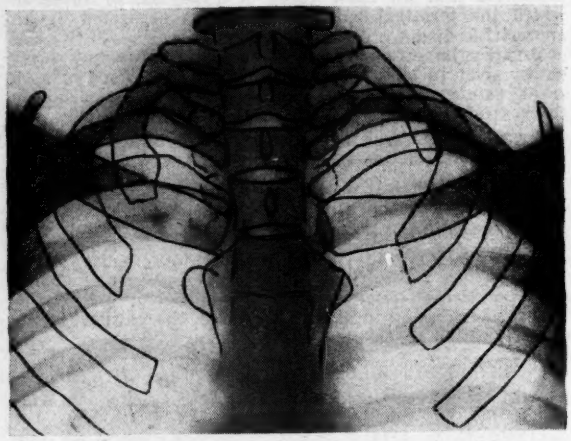

FIGURE IIB.

Line drawing made from radiograph to show the arrangement of the bony structures more clearly.

Cold Immersion Test.[2]—When the hands were immersed for ten minutes in water at 15° to 17° C., the tips of the fingers of the left hand became slightly cyanosed.

Reactive Hyperæmia Test.[2]—The hands were immersed in water at 35° C. for ten minutes, taken out and elevated above the head, and cuffs were inflated to occlude the arteries in the arm; the hands were returned to the water for five minutes, and then each cuff was deflated rapidly in turn. The flush of reactive hyperæmia reached the tips of the fingers of the right hand in less than three seconds; in the left hand it took from thirty to sixty seconds.

Hot Immersion Test.[2]—After immersion of the hands in water at 45° C. for ten minutes, pulses were felt at the bases of all fingers of the right hand, and capillary pulsation was seen in all nail beds. No arterial pulses could be felt in the left hand, and faint capillary pulsation was seen in one nail bed only.

Calorimetry.[3]—The resting heat elimination from his left hand was 16 calories per 100 millilitres per minute, with no increase from immersing the other hand and forearm in water at 44° C. ("reflex" hyperæmia test).

Surgical Treatment and Findings.

The patient was readmitted to hospital in June, 1953, four months after the sympathectomy, and Dr. H. C. Trumble explored the left side of the neck, divided the scalene muscles which were compressing the subclavian

[1] Adson's manœuvre: the patient sits with the arms abducted horizontally and the forearms perpendicular. The physician palpates the pulses while the patient is instructed to turn his face towards the side being examined, and to take a deep breath and hold it while the neck is extended. (Adson and Coffey, 1927.)

[2] These tests are modified from those described by Lewis and Pickering (1933).

[3] See Barnett and Wigley (1953).

artery proximal to the aneurysm, and divided the first rib. The following is a summary of the operation notes.

> An incision was made a little above and parallel to the clavicle. A good deal of the clavicular portion of the sterno-mastoid muscle was divided. The *scalenus anterior* muscle, brachial plexus, and a spindle-shaped aneurysmal dilatation of the subclavian artery lying lateral to the *scalenus anterior* muscle were displayed. An attempt was made by pressing the shoulder backwards to see if the clavicle could have been responsible for the compression of the subclavian artery, but as far as could be ascertained this was not so. The *scalenus anterior* muscle was cut through just above its insertion until only a few of the most posterior tendinous fibres remained. These appeared definitely to compress the subclavian artery. When the band was lifted off the artery by a hook the vessel expanded, and when allowed to fall back it pressed into the vessel. This effect was exaggerated by inspiration. The muscle was then completely divided. The *scalenus medius* muscle was then divided near its insertion, and the first rub was cut through posterior to the brachial plexus in order to allow the anterior segment to subside. There appeared to be a tubercle on the rib at the anterior part of the insertion of the *scalenus medius* muscle, and this could have embarrassed the subclavian artery. The spindle shaped aneurysmal dilatation appeared to contain a good deal of clot, but it still pulsated. The wound was closed *secundum artem*.

The relation of the structures in the region of the subclavian aneurysm as interpreted from the findings at operation is shown in Figure IV.

One week later, the right side of the patient's neck was explored. The *scalenus anterior* muscle was cleared. It did not appear to be causing any embarrassment to the subclavian artery; however, it was divided a little above its insertion. The artery appeared to be of normal calibre behind the muscle and lateral to it.

Further Progress.

There were no post-operative complications. Since the operation the circulation in the left hand has improved; in fact, this hand has become warmer than the right. There have been no further spontaneous attacks of pallor, although if the hands are held above the head and clenched a few times the left hand becomes much paler than the right. Pulsation in the left brachial artery is still present to the middle of the humerus only. A very faint pulsation has occasionally been detected in the radial artery.

In February, 1954, eight months after operation, both hands were warm and pink. The left hand was dry, and the fingers of this hand were more highly coloured than on the right.

Grip Test.[1]—Pain in the hand was produced after 59 grips (average of three runs).

Cold Immersion Test.—The fingers of the right hand remained pink; those of the left hand became slightly cyanosed.

Reactive Hyperæmia Test.—The fingers of the right hand flushed within three seconds; those of the left hand within ten seconds.

Hot Immersion Test.—Capillary pulsation was noted in the nail beds of all fingers of both hands. Arterial pulses (weak) were detected at the bases of the digits of the left hand, with the exception of those at the base of the thumb and the ulnar side of the little finger.

Calorimetry.—The resting heat elimination from his left hand was 30 calories per 100 millilitres per minute. There was no increase from the "reflex hyperæmia" test.

Discussion.

This patient's history has many points of interest, and three of these have been chosen for brief discussion: the primary structural abnormality, the cause of the vascular symptoms and the mechanism of the aneurysm formation.

[1] This and succeeding tests were performed in the manner previously described in this paper.

Primary Structural Abnormality.

The most widely recognized structural abnormality in the thoracic outlet region associated with vascular symptoms is a cervical rib. This has been long recognized, and has been fully discussed by various authors. The first clinical description was given by Cooper (1818). The earliest cases of vascular complication of a cervical rib explored at operation were published by Verneuil in 1855 and by Coote in 1861. A detailed description of the symptomatology, diagnosis and surgical treatment of cervical ribs was written by Keen in 1907. A classical

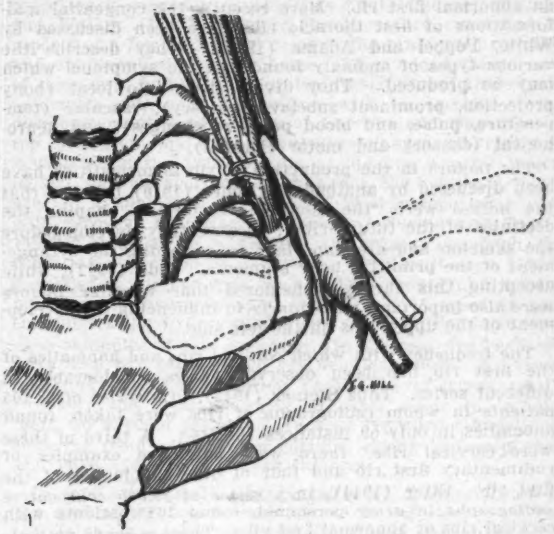

FIGURE IV.

Arrangement of structures in the region of the subclavian aneurysm as deduced from the findings at operation.

paper on the subject of the vascular complications of cervical ribs and first thoracic rib abnormalities was published by Eden in 1937. He described the case previously reported by Lewis and Pickering and two further cases of his own of vascular disturbance from abnormality at the thoracic outlet (one patient with cervical rib and the other with costo-clavicular compression) and collected 45 other cases from the literature.

The importance of pressure by either a normal or an abnormal first thoracic rib in the production of symptoms was recognized later than that of a cervical rib. Bramwell (1903) was the first to suggest that pressure from a normal first rib could produce symptoms—by pressure on the first dorsal nerve root. Thomas Murphy[1] (1910) of Melbourne was the first to resect a "normal" first dorsal rib for relief of symptoms. Pressure from a normal first rib was discussed by Brickner (1927), who quoted several instances in which a normal first rib had been resected for brachial plexus pressure by other workers, and described similar cases of his own. Symptoms were mainly neurological. One patient had a cold, dusky, sweating hand with œdema of the limb; symptoms were relieved by resection of a normal first rib.

Congenital abnormalities of the first rib were first described by Hunauld in 1740. Keen (1907) first recognized that they could produce symptoms. The radiological appearances in one of Keen's cases bear a resemblance to our own.

[1] Murphy referred to his patient having an "apparently normal" first rib. The radiographs produced in his article show that the rib was projecting abnormally outwards. The symptoms were mainly nervous (pain, weakness, and sensory changes); but there was also some vascular disturbance in the form of "dusky blue discolouration of wrist and hand".

> On the left side is a still more peculiar condition. The first rib, instead of curving forward from the spine, as does the right, changes its direction at its articulation with the transverse process and thence runs almost in a straight line downward and outward. The tip of it is lost at the point where the first rib and the clavicle cross each other. From that point to the sternum there is no rib . . .

In the same year, Russel reported a case of bilateral vascular disturbance in the hands, including gangrene of the right finger tips, associated with rudimentary first thoracic ribs. Autopsy showed thrombosis of both subclavian arteries. Brickner and Milch (1925) discussed the simulation of the neurological features of a cervical rib by an abnormal first rib. More recently the congenital malformations of first thoracic ribs have been discussed by White, Poppel and Adams (1945). They describe the various types of anomaly found and the symptoms which may be produced. They divide these into local (bony projection, prominent subclavian artery), vascular (temperature, pulse, and blood pressure changes), and neurological (sensory and motor changes).

The factors in the production of rib abnormalities have been discussed by anatomists. Jones (1910) believed that the nerves were "the dominant factors in shaping the destinies of the future ribs", because they develop before the skeleton and are important in governing the arrangement of the primitive body segments. Todd (1912), while accepting this theory, considered that vascular factors were also important, particularly in influencing the development of the upper ribs on the left side.

The frequency with which cervical ribs and anomalies of the first rib has been observed varies considerably in different series. Thus Steiner (1943), in a series of 38,105 patients in whom radiographs of ribs were taken, found anomalies in only 59 instances (0·15%). A third of these were cervical ribs; there were only five examples of rudimentary first rib and four of other anomalies of the first rib. Etter (1944), in a series of 40,000 consecutive radiographs in army personnel, found 261[1] patients with cervical ribs or abnormal first ribs. There were 68 patients (0·17% of the total) with cervical ribs. Of the 193 patients (0·44%) with abnormalities of the first thoracic ribs, 31 (0·08%) had "anomalous" first ribs (appearance of healed fracture or pseudo-arthrosis in the mid-portion), 67 (0·17%) had rudimentary first ribs, 79 (0·20%) had synostosis of first and second ribs, and 16 (0·04%) had bifid first ribs.

It is a reasonable supposition that our patient's symptoms were related to his abnormally short rib, which allowed less room than normal for insertion of the scalene muscles, and that the brachial artery was thus more readily compressed between them.

Cause of Symptoms.

The main questions concerning the mechanism of the symptoms in thoracic outlet syndromes are: (*a*) What structures are causing compression? (*b*) What structures are compressed? (*c*) How does this compression produce symptoms?

It was at first thought that in patients with cervical rib associated with vascular symptoms the rib itself was causing the compression. Adson and Coffey (1927) emphasized the important part played by the *scalenus anterior* muscle in these cases. Naffziger believed that in certain instances this muscle was primarily at fault and could cause symptoms in the absence of a cervical rib. Ochsner, de Bakey and Gage (1935) attributed symptoms to "spasm" of this muscle. In our patient it was shown at operation that the artery was compressed by the *scalenus anterior* muscle; this was not due to spasm, but resulted from the proximity of its insertion to that of the *scalenus medius* muscle due to the shortness of the first rib.

Symptoms from thoracic outlet anomalies are usually divided into vascular and nervous types (Walshe, 1951); these features often occur separately. Various explanations of the production of the vascular symptoms have been given in the past. One view was that they were due to an irritation of sympathetic nerves (Gordon, 1901; Todd, 1912; Telford and Stopford, 1931). Lewis and Pickering (1934) produced a convincing argument that in the case reported by them the symptoms were directly due to affection of the subclavian artery, resulting in thrombosis and embolism. In Eden's (1939) two cases, aneurysm of the subclavian artery was demonstrated at operation; arterial occlusion had occurred in one case before and the other after operation, and it is highly probable that the symptoms were produced in the manner postulated by Lewis and Pickering. Walshe and his co-workers (1944) reported a number of cases of cervical rib, rudimentary and otherwise abnormal first ribs; they also believed, on clinical and anatomical grounds, that the vascular symptoms were due to pressure on the artery and not due to sympathetic nervous irritation. This view has been endorsed by White and his co-workers (1945). Our case is particularly complete. The history indicated repeated occlusions of vessels of the hand, then of the radial artery and finally of the lower part of the brachial artery. At operation, thrombus was felt in the aneurysm. There was no evidence of involvement of nerves.

Mechanism of Aneurysm Formation.

We shall terminate this discussion with a short comment on the possible mechanism of production of the aneurysm of the subclavian artery. Dilatation of the third part of the artery associated with compression of the second part seems paradoxical, as the mean pressure beyond an obstruction is reduced. A similar phenomenon occurs in other regions distal to a vascular constriction, as in the post-stenotic dilatation in certain instances of pulmonary stenosis and coarctation of the aorta. Halsted (1916), after many failures, produced aneurysms in the aorta by constriction with metal bands. The degree of constriction was critical—to one-quarter to one-third of the diameter. Reid (1916) examined these aneurysms and found that the amount of elastic tissue was reduced. Halsted suggested that the abnormal "whirlpool-like play of blood" in the relatively dead pocket just below the site of constriction, and the lowered pulse pressure might be the chief factors concerned in the production of the dilatations. Cohen (1952) has suggested that a pulsatile blood flow is necessary for the health of elastic tissue; when the pulsation is greatly reduced, as when it is distal to a constriction, elastic tissue degenerates and aneurysmal dilatation occurs. This does not seem a reasonable explanation, as it would not explain why the dilatation should occur only just distal to the constriction. Recently a physicist (Mr. B. McA. Sayers) has drawn our attention to the fact that when turbulence is set up distal to a constriction, although the average pressure is reduced, the instantaneous pressures may be tremendous. (This mechanism is responsible for the pitting of propellors of ships.) It seems feasible that the rapidly changing pressure may be responsible for disruption of elastic fibres of the vessel wall, and this may lead to aneurysmal dilatation.

Summary.

A case of subclavian aneurysm associated with a short "first" rib is reported. There was definite evidence of constriction of the artery by the *scalenus anterior* muscle proximal to the aneurysm.

The history indicated repeated embolic occlusions of arteries of the arms, and at operation the aneurysm was found to contain clot. Thus support is given to the view of Lewis and Pickering and of others that vascular disturbance in the hands due to abnormalities at the thoracic outlet may be caused by embolism from thrombus in the subclavian artery.

The mechanism of aneurysm formation is considered. It is suggested that it is due to disruption of elastic tissue by rapidly changing pressures occurring in turbulent flow.

[1] This figure is obtained by our own addition of the numbers in various groups given by the author. The percentages are our calculations.

Acknowledgements.

Our thanks are due to Dr. H. C. Trumble for the operation report and advice in the writing of this paper, to Dr. B. L. Deans for the radiographic examination and discussion of the findings, and to Dr. T. E. Lowe for helpful criticism. The photograph (Figure I) was produced by Mr. T. O'Connor. The line drawing (Figure IV) was made by Miss I. Hill.

References.

ADSON, A. W., and COFFEY, J. R. (1927), "Cervical Rib: A Method of Anterior Approach for Relief of Symptoms by Division of the Scalenus Anticus", *Ann. Surg.*, 85: 839.

BARNETT, A. J., and WIGLEY, G. R. (1953), "Calorimetry: A Method of Estimating Peripheral Blood Flow", M. J. AUSTRALIA, 2: 236.

BRAMWELL, E. (1903), "Lesion of the First Dorsal Nerve Root", *Rev. Neurol. & Psychiat.*, 1: 236.

BRICKNER, W. M. (1927), "Brachial Plexus Pressure by the Normal First Rib", *Ann. Surg.*, 85: 858.

BRICKNER, W. M., and MILCH, H. (1925), "First Dorsal Simulating Cervical Rib—by Maldevelopment or by Pressure Symptoms", *Surg., Gynec. & Obstet.*, 40: 38.

COHEN, S. (1952), "Peripheral Aneurysm and Arterio-venous Fistula", *Ann. Roy. Coll. Surg. Eng.*, 11: 1.

COOPER, A. (1818), quoted by *Adson, A. W., and Coffey, J. R., loco citato.*

COOTE, F. (1861), *Lancet*, 1: 360; quoted by Eden, K. C., *loco citato.*

EDEN, K. C. (1939), "The Vascular Complications of Cervical Ribs and First Rib Abnormalities", *Brit. J. Surg.*, 27: 111.

ETTER, L. E. (1944), "Osseous Abnormalities of the Thoracic Cage Seen in Forty Thousand Consecutive Chest Photoroentgenograms", *Am. J. Roentgenol.*, 51: 359.

GORDON, T. E. (1901), "A Case of Cervical Rib: Obliteration of the Arteries of the Right Arm: Gangrene of a Portion of Several Fingers: Excision of the Rib", *Brit. M. J.*, 1: 1395.

HALSTED, W. S. (1916), "An Experimental Study of Circumscribed Dilatation of an Artery Immediately Distal to a Partially Occluding Band, and its Bearing on the Dilatation of the Subclavian Artery Observed in Certain Cases of Cervical Rib", *J. Exper. Med.*, 24: 271.

HUNAULD (1740), Communication to the Royal Academy of Sciences in 1740, published in Amsterdam, 1744; quoted by White, J. C., Poppel, M. H., and Adams, R., *loco citato.*

JONES, F. WOOD (1910), "On the Relation of the Limb Plexuses to the Ribs and Vertebral Column", *J. Anat. & Physiol.*, 44: 377.

KEEN, W. N. (1907), "The Symptomatology, Diagnosis and Surgical Treatment of Cervical Ribs", *Am. J. M. Sc.*, 133: 173.

LEWIS, T., and PICKERING, G. W. (1934), "Observations upon Maladies in Which Blood Supply to Digits Ceases Intermittently or Permanently, and upon Bilateral Gangrene of Digits; Observations Relevant to So-called Raynaud's Disease", *Clin. Sc.*, 1: 354.

MURPHY, T. (1910), "Brachial Neuritis Caused by Pressure of First Rib", M. J. AUSTRALIA, 15: 582.

NAFFZIGER, H., quoted by Ochsner, A., Gage, M., and de Bakey, M. (1935), *loco citato.*

OCHSNER, A., GAGE, M., and DE BAKEY, M. (1935), "Scalenus Anticus (Naffziger) Syndrome", *Am. J. Surg.*, 28: 669.

REID, M. R. (1916), "Partial Occlusion of the Aorta with the Metallic Band. Observations on Blood Pressure and Changes in Arterial Walls", *J. Exper. Med.*, 24: 287.

RUSSEL, C. K. (1907), *M. Rec.*, 71: 253; quoted by Eden, K. C., *loco citato.*

SAYERS, B. MCA. (1953), personal communication.

STEINER, H. A. (1943), "Roentgenologic Manifestations and Clinical Symptoms of Rib Abnormalities", *Radiology*, 40: 175.

TELFORD, E. D., and STOPFORD, J. S. B. (1931), "The Vascular Complications of Cervical Rib", *Brit. J. Surg.*, 18: 557.

TODD, T. W. (1912), "The Vascular Symptoms in 'Cervical' Rib", *Lancet*, 2: 362.

TODD, T. W. (1912), *Anat. Anz.*, 41: 257 and 335; quoted by White, J. C., Poppel, M. D., and Adams, R., *loco citato.*

VERNEUIL, A. A. (1855), quoted by Baumgartner, A., Clerc, A., and Macrez, C. (1938), *"Sur l'anévrysme artériel de voisinage et la gangrène ischémique des doigts en rapport avec les côtes cervicales. Leur traitement chirurgical", Presse méd.*, 2: 1665.

WALSHE, F. M. R. (1951), "Nervous and Vascular Pressure Syndromes of the Thoracic Inlet and Cervico-axillary Canal", in Feiling A. (1951), "Modern Trends in Neurology", Butterworth, 542.

WALSHE, F. M. R., JACKSON, H., and WYBURN-MASON, R. (1944), "On Some Pressure Effects Associated with Cervical and with Rudimentary and 'Normal' First Ribs, and the Factors Entering into their Causation", *Brain*, 67: 141.

WHITE, J. C., POPPELL, M. H., and ADAMS, R. (1945), "Congenital Malformations of the First Thoracic Rib", *Surg., Gynec. & Obst.*, 81: 643.

POSTERIOR FOSSA CYSTS DUE TO CONGENITAL ATRESIA OF THE FORAMINA OF LUSCHKA AND MAGENDIE.[1]

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VERY few instances of posterior fossa cysts due to congenital atresia of the foramina of Luschka and Magendie have been recorded in the medical literature, and the condition is generally spoken of as a rare disorder. Taggart and Walker (1942) reviewed the literature consisting of reports of a few undetailed cases of the nineteenth century, and six twentieth century cases. They added three cases of their own, and gave a detailed description of these cysts and their pathogenesis, together with an account of the development of the foramina of Luschka and Magendie, of the cerebellum, and of the transverse sinuses. Shyrock and Alexander in the 1943 *Bulletin of the Los Angeles Neurological Society* reported a further case, the details of which are not available to us. In 1944, Walker reported a case with recovery following operation. Coleman and Toland (1948) reported an additional two cases, in which the condition was cured by operation. Dorothy Russell (1949) described a further case in her 1949 monograph on the pathology of hydrocephalus. Six out of 11 cases of non-communicating hydrocephalus described by Benda in 1952 were examples of this condition. In the past five years we have seen five cases which are detailed below. We therefore feel that the condition cannot be of great rarity. Probably the great majority of cases occur in infants. Many so affected perhaps have been regarded clinically as suffering from hopeless non-communicating hydrocephalus, and have excited no further interest.

In this developmental abnormality no opening forms in the roof of the fourth ventricle at its inferior end. The lateral recesses may also remain closed, and an unusual variety of non-communicating hydrocephalus results. Reference to Figure I will show that at the fourth month of fœtal life, the cerebellum is a dumb-bell shaped structure, the crossbar being the vermis, which lies above and anteriorly. It is at this stage of development that distension of the fourth ventricle prevents further growth of the vermis in the postero-inferior direction. There results a cystic expansion of the fourth ventricle, into the lateral walls of the cyst bulge the postero-medial aspects of the cerebellar hemispheres, and the posterior wall of the cyst is membranous, being composed of a vascular translucent membrane formed from the ependymal lining of the roof of the ventricle and the vascular lepto-meninges. Anteriorly, in the superior wall, is the incompletely developed vermis. A failure of descent of the transverse sinuses is a frequent associated feature. At the third month of fœtal life the *torcular Herophili* lies in the mid-line, just above the lambda. The dilatation of the fourth ventricle hinders the normal folding backwards of the cerebral hemispheres which carries the sinuses backwards and downwards to their normal position on the occipital bone. They therefore retain their earlier position on the inferior part of the parietal bone. Because their impression is moulded on the calvarium, it is frequently possible to diagnose this condition from a careful examination of plain X-ray films of the skull, especially in older children and adults. One would think that such a complete closure of the ventricular system would result in a rapidly expanding non-communicating destructive hydrocephalus, incompatible with prolonged useful life. Such is not necessarily the case. As will be seen, there are several examples of this anomaly occurring in fairly normal older children and adults. Taggart and Walker (1942) make the following statement:

[1] A summary of this paper was read at the annual meeting of the Australian Pædiatric Association, Canberra, April 8 to 11, 1954.

Several factors operate in cases of this anomaly to produce a balance of cerebro-spinal fluid dynamic pressures. Certainly the abnormally small choroid plexuses produce less ventricular fluid than normal. Again, the cyst wall probably acts as a semi-permeable membrane permitting dialysis of the ventricular fluid, which has a lower osmotic pressure than cerebro-spinal fluid in the basal cisterns and the subarachnoid space. A third factor is the absorption of some fluid by the numerous vessels in the cyst wall itself. These compensating mechanisms probably have little reserve, so that a slight disturbance of any one of them might precipitate a state of intracranial hypertension. It is reasonable to suppose that in those cases in which the foramens open late or inadequately, development may be normal until some extrinsic factor upsets the delicate balance of the cerebro-spinal fluid dynamic pressure.

In clinical histories, trauma has frequently been such a precipitating factor. Sometimes cases are encountered in which, whilst the foramen of Magendie has remained closed, the foramina of Luschka are patent. These children are probably more likely to develop fairly normally, save for a variable degree of hydrocephalus.

Clinical Records.

CASE I.—This male infant appeared to be normal at birth, but an enlargement of the head was noted at the age of three weeks, and at seven weeks his head circumference was 19 inches. At this time a ventricular puncture was performed, and two mils of phenolsulphonphthalein were injected into the lateral ventricle; the child was then sat up and a lumbar puncture was performed. The dye appeared in three minutes. Twenty-five millilitres of cerebro-spinal fluid were removed and replaced with oxygen; the flow then ceased, and the oxygen bubbled back. X-ray films of the skull showed that no oxygen had entered the ventricles. The skull was grossly enlarged, with considerable widening of the sutures. The position of the transverse sinuses could not be determined. The cerebro-spinal fluid contained eight cells and 70 milligrammes of protein per 100 millilitres. By the age of three months the hydrocephalus had progressed and the head circumference was 20·3 inches. Because of the reasonably rapid transit of dye from the ventricles to the lumbar subarachnoid space, it was thought that some communication must be present between the ventricles and the subarachnoid space, despite the non-entry of the air into the ventricles. Accordingly a uretero-arachnoid anastomosis was performed. No relief of the hydrocephalus was obtained until about four weeks after the operation, when there occurred a sudden collapse of the anterior fontanelle, overriding of the skull bones, and the passage of much urine, resulting in acute dehydration which was countered with much difficulty. The child survived for a further three months, and finally died in an acute episode of dehydration at the age of eight months.

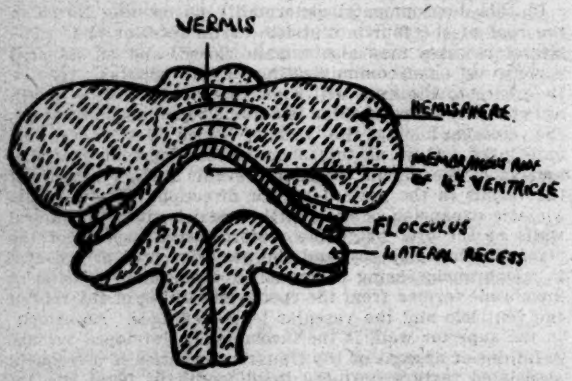

FIGURE I.
Dorsal aspect of cerebellum and roof of fourth ventricle in a 120 millimetre (fourth month) embryo. (From Hamilton, Boyd and Mossman, 1946.)

At autopsy, it was found that the posterior cranial fossa was greatly enlarged owing to an upward displacement to the level of the lambda of the tentorium and its enclosed sinuses. This enlarged fossa was occupied by an enormous, centrally placed, thin-walled cyst, which had displaced the cerebellar hemispheres laterally so that they were widely separated, and no posterior vermis was evident (Figure II). The posterior wall of the cyst was composed of a thin vascular and translucent membrane, through which the floor of the fourth ventricle was clearly visible (the wall of the cyst being the roof of the fourth ventricle). As the membrane was traced upwards and forwards beneath the tentorium (Figure III), a diverticulum was found to pass upwards through the tentorial opening to become loosely attached to the arachnoid on the medial surface of the cerebral hemispheres. The membrane then became continuous with the inferior surface of the "anterior" vermis (in reality the incompletely developed and displaced total vermis). Laterally (Figure IV) the membrane passed forwards and appeared to be attached to the lateral margins of the cerebellar hemispheres, which thus seemed to be within the cyst cavity and to form its antero-lateral walls. However, more detailed examination showed that the membranous cyst wall continued medially on the surface of the cerebellar hemispheres to become continuous with the ependyma of the floor of the fourth ventricle. In general, however, the membrane covering the posterior and medial aspect of the cerebellar hemispheres was adherent to the pia-arachnoid covering the cerebellum, and could be separated from it only with difficulty. The lateral recesses did not open into the subarachnoid space, but ended in small cystic expansions anteriorly. One such lateral recess, with its expanded cystic extremity, is shown in Figure V. The wall of the large cyst was complete posteriorly save for a small tear, which probably occurred at the time of the collapse of the head three months before death. There was thus a complete absence of the foramina of Luschka and Magendie. Choroid plexuses could not be recognized within the cyst on gross inspection; but one small atropic plexus was found microscopically.

FIGURE II.
Interior of the cyst from Case I. The significant features are marked.

A considerable overlapping of the bones of the cranial vault at the sutures left no doubt that a profound reduction in volume of the cranial contents had occurred, because of the rupture of the cyst membrane coupled with the uretero-arachnoid anastomosis. The aqueduct and the third and lateral ventricles were still considerably dilated, and gross general hydrocephalus was thus present. There was also an absence of the *corpus callosum*. Microscopic examination of the closed cystic expansions in which the lateral recesses

ended anteriorly (Figure V) revealed that these small cysts were lined completely by a layer of ependymal cells directly continuous with those lining the lateral recess. External to this ependymal lining was a clearly defined layer of fibrous glia, which was relatively thick near the mouth of the lateral recess, but had become considerably attenuated over the apex of the cyst. Outside this again was a thin layer of pia-arachnoid containing a few inflammatory cells. This microscopic study, therefore, served to demonstrate without any question the closed nature of the lateral recess.

CASE II.—The subject was a premature female child, born at thirty-three weeks' gestation after a threatened miscarriage at five weeks. Deformities of both hands and the right foot were evident at birth, and an enlarged head was noted at the age of two weeks. At six weeks the head circumference was 15 inches, the anterior fontanelle was wide and full, and the sutures were open. A pneumoencephalogram was performed by the lumbar route. The flow of cerebro-spinal fluid was slow, with a tendency for the oxygen to bubble back, and only 18 millilitres could be inserted. The cerebro-spinal fluid protein content was 60 milligrammes per 100 millilitres. X-ray films of the skull showed that only a little oxygen was in the lateral ventricles, which, though poorly filled, could be seen to be enlarged, and to have an upward peaking of their dorsal angles somewhat suggestive of agenesis of the *corpus callosum*. No signs of the transverse sinus could be detected in the X-ray films.

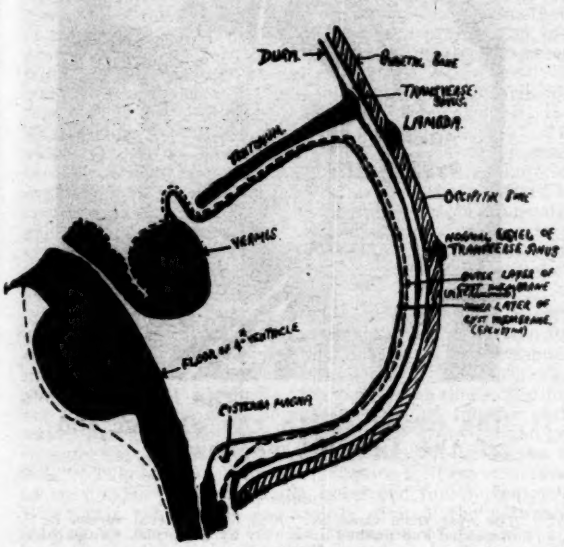

FIGURE III.
Diagram of vertical section through cyst, Case I.

At the age of eight months the child appeared hydrocephalic. The head circumference was 18·75 inches and the anterior fontanelle was enlarged and bulging. A plain X-ray film of the skull showed considerable increase of convolutional markings. No marking indicating the position of the transverse sinus could be seen. A further pneumoencephalogram confirmed the diagnosis of a largely non-communicating hydrocephalus, and a posterior fossa exploration was undertaken. At operation, deep to the dura, a large cyst was found. One could look through the thin translucent roof directly into the fourth ventricle, and the cerebellar vermis could be seen high up anteriorly. A portion of the roof was removed and the wound was closed. Death occurred two days later.

The autopsy findings were very nearly identical with those in Case I. In this case, too, the lateral recesses appeared to be closed, though this was not verified histologically. In this case also the *corpus callosum* had failed to develop.

CASE III.—The subject, a boy, was examined at the age of five months, when he was suffering from hydrocephalus. A cystic swelling had been removed from the back of his head at the age of seven weeks. It did not communicate with the intracranial contents. A lumbar pneumoencephalogram was taken after dye had been injected into the ventricles. The passage of the dye was delayed, no colour being detected in the lumbar cerebro-spinal fluid for eight minutes. X-ray films (see Figure VI) showed good filling of the ventricles, which were moderately dilated. A finger-like process of air extended backwards from the region of the right lateral ventricle to the mid-line posteriorly. (At the time it was thought that this was probably connected with the cyst previously removed; it was later realized that this was subtentorial air at a much higher level than normal.) The basal cisterns were well filled and the cortical markings numerous and large. No abnormality was detected in the cerebro-spinal fluid. The position of the transverse sinus was not noted. (Later review of these X-ray films showed that it could be detected and was abnormally high.) As the site of the obstruction causing the hydrocephalus was obscure, a ventriculogram was taken five weeks later, 40 millilitres of cerebro-spinal fluid being removed and replaced with oxygen. The X-ray films (see Figures VII and VIII) showed good filling of the hydrocephalic ventricles, which communicated with a large cyst in the posterior fossa. The posterior and inferior horns of the lateral ventricles were displaced upwards and forwards. The child reacted badly to the disturbance caused by ventriculography and died two days later.

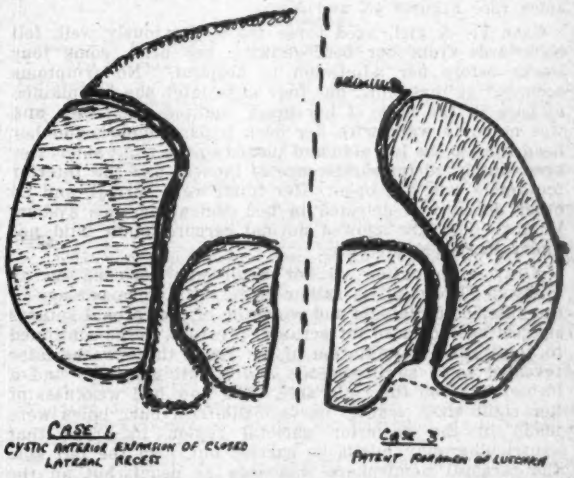

FIGURE IV.
Diagram of transverse section through cysts in Cases I and III at level of lateral recess.

At autopsy there were certain significant differences between this cyst and the previous two. The transverse sinus was high, as before, but the cyst was much smaller, and the reflection of the membranous wall of the cyst from the cerebellum to the dura occurred along a line placed much more medially (see Figure IV). The lateral recesses appeared to open freely into the subarachnoid space anteriorly (the point of a probe passed along the lateral recess could be freely moved about in the subarachnoid space anteriorly). This possible patency of the foramina of Luschka was not verified histologically, and on subsequent review of the specimen it was found then not to be suitable for this type of examination.

CASE IV.—A girl was examined at the age of seven months, when she had been vomiting for four weeks and was irritable and drowsy. Previously quite well, she had

made good progress. She had sat up at five months, and could say a few words. She was noted to have a large head (19·75 inches in circumference) with a bulging anterior fontanelle and separated sutures. Moderate neck and spine stiffness was present. No marking of the transverse sinuses could be detected in the X-ray films of her skull. Her ventricles were decompressed by repeated ventricular taps, and her condition improved. Her cerebro-spinal fluid was normal. Ventriculography was performed. A distended fourth ventricle was visualized, and some of the air had come out of the ventricles into the basal cisterns, suggesting that the hydrocephalus was a communicating one. To confirm this, a week later dye was injected into the left lateral ventricle; she was then sat up, and lumbar puncture was performed. Dye did not appear in the lumbar cerebro-spinal fluid for six minutes. Thirty-five millilitres of cerebro-spinal fluid were then removed and replaced with oxygen. The flow of cerebro-spinal fluid was slow and ceased at 35 millilitres. X-ray films showed that no air had entered the ventricles. It was accordingly thought that the hydrocephalus was largely non-communicating, and a posterior fossa exploration was made two weeks later. At operation, when the dura was opened a large cyst was exposed. Portion of the membranous roof of the cyst was removed and the wound was closed. A cerebro-spinal fluid leak occurred on the ninth post-operative day, and she developed meningitis and died four weeks later.

At autopsy it was found that the tentorium and its enclosed sinuses were at their normal level, and the posterior cranial fossa was not enlarged in size. The cyst was much smaller than the others, there being only moderate mid-line separation of the cerebellar hemispheres, and the vermis was only a little reduced in thickness. Well formed, but closed, lateral recesses were present on both sides (see Figures IX and X).

CASE V.—A girl, aged three years, previously well, fell backwards from her bed, striking her head, some four weeks before her admission to hospital. No symptoms occurred at that time, but four days later she complained of pain in the back of her head, vomited every day, and was noted to walk with her back hyperextended, and her head bent to the left side and turned to the right side. Her head was large (circumference 21 inches) and the anterior fontanelle was still open. Her fundi were normal, and no other defect was detected in her central nervous system. Lumbar puncture showed normal cerebro-spinal fluid not under increased pressure.

X-ray examination of her skull was reported on as follows: "The skull is rather large, with some patency of the anterior fontanelle and widening of the coronal sutures suggesting increased intracranial pressure." She continued to vomit, and examination of her fundi three weeks later revealed some swelling (one and a half diopters) and a hæmorrhage on the right side. She now had weakness of her right sixth cranial nerve. Bilateral burr holes were made in the posterior parietal region in order that ventriculography might be carried out. On the left side the cerebral hemisphere was seen as usual, but on the right side a large cyst was entered. It was thought to be a subdural hygroma, and was drained. Cerebro-spinal fluid continued to leak away after removal of the tube, resulting in gross dehydration and death twelve days later.

At autopsy it was found that the supposed subdural hygroma was a large posterior fossa cyst, that the tentorium was raised to above the level of the lambda, and that the left burr hole had been placed above the tentorium and the right below the tentorium, the cyst being thus entered; the cyst was similar in appearance to those in Cases I and II. Unfortunately the state of the lateral recesses and foramina of Luschka was not noted. Review of the patient's X-ray films revealed clear evidence of high attachment of her tentorium (Figure XI). (This was the first cyst encountered by us, but is described last for convenience of description.)

Discussion.

Analysis of the 24 cases available to us gives the following information.

Thirteen subjects were infants, five were aged from three to six years, and six were aged from thirteen to fifty-nine years.

Of the eleven older patients, five were fairly normal mentally, six were greatly retarded.

Thirteen of the subjects were females and seven males.

The foramen of Magendie was closed in all cases.

The foramina of Luschka were closed in 16 cases and patent in three cases, and the condition was unstated in five cases. In the 16 subjects with closed foramina, the position of the transverse sinus was high in five, normal in three and unstated in eight. Of the three subjects with patent foramina of Luschka, the position of the transverse sinus was high in one, normal in one, and unstated in one.

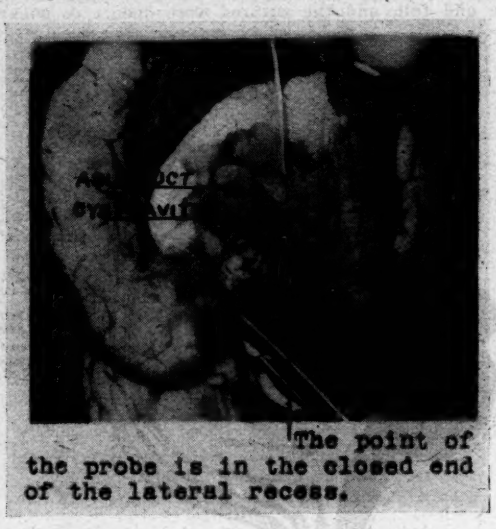

FIGURE IX.
The cyst from Case IV. The blind lateral recess is distended and pushed anteriorly by the probe. Compare Figure X.

The position of the transverse sinus was stated in 13 cases. In nine it was high, in four it was normal. In the nine cases in which it was high, the foramina of Luschka were closed in five and open in one (our Case III), and the condition was unstated in three. In the four subjects in which the position was normal, the foramina of Luschka were closed in three and open in one.

Operation was performed in 15 cases, with 10 deaths and five recoveries. The successful operation was a fourth ventriculostomy, in which the inferior portion of the membranous cyst wall is removed in order to allow the ventricular fluid to drain into the cisternal subarachnoid space.

It is of interest that in our Cases I and II, absence of the *corpus callosum* was an associated abnormality. Bunts and Chaffee (1944) state that this structure develops between the third and fifth month. Taggart and Walker (1942) make the following statement:

> The time at which the foramen of Magendie opens has not been definitely established for man. Hess found the foramen patent in a twelve centimetre (fourth to fifth month) human embryo, and Retzius stated that it was open at the beginning of the fourth month. Kölliger stated that the thinning of the caudal wall of the fourth ventricle which initiates the process of forming the foramen, begins at the fourth month of embryonic life . . . . The foramens of Luschka are formed by a thinning of the epithelium (Folco) in the lateral recesses of the fourth ventricle; according to Blake, this thinning becomes apparent about the eighth week of fœtal life. The foramen is patent at the end

of the fourth month (Blake, Hess, Retzius). Karlefors, however, stated that the thinning of the recess wall occurs in the fifth month and the foramen is open at the end of the sixth month.

In Cases I, II and IV we experienced difficulty in determining whether the hydrocephalus was communicating or non-communicating. For example, in Case I the dye test led to a belief of some communication, whereas the failure of filling of the ventricles in the pneumoencephalogram was against communication. In Case II some little air actually entered the ventricles, despite the fact that at autopsy the hydrocephalus appeared completely sealed off from any communication with the subarachnoid space. Are both these phenomena to be explained by the permeability of the cyst membrane?

From the examination of our specimens at operation and at autopsy, and by the microscopic examination of serial sections in Cases I and IV, we are of the opinion that it is very difficult to determine the state of patency of the foramina of Luschka in these cases by the first two methods of examination. We had considered that if the point of a probe which had been passed through the lateral recess from the fourth ventricle could be moved about freely in what seemed to be the subarachnoid space anteriorly, the foramen of Luschka must be patent. For example, this was thought to be so in Case IV (see Figure IX). The value of this test may be very doubtful in these cases, because the lateral recess may end blindly in an anterior cystic expansion roofed over by a thin membrane formed by ependyma-lined glia internally and pia-arachnoid externally, and the free movement of the probe within this cyst is very deceptive (see Figures V and X). It would therefore appear that only serial sections can safely be employed to answer this point.

In Case III, air inserted by lumbar puncture readily entered the ventricles (see Figure VI). It is generally considered that the foramina of Luschka have no function in the filling of the ventricles with air from below. Davidoff and Dyke (1946) state that "the mouths of these openings are located at the ends of narrow tortuous tubular pathways, thereby presenting mechanical difficulties for the gas to enter, although no obstruction exists to the egress of fluid". This statement, however, applies to these structures as they exist in the normally developed fourth ventricle. When this ventricle is enormously dilated, the pathways leading to the mouths of the foramina of Luschka are somewhat less narrow and tortuous than is their normal state. Robertson (1941) states that "the foramina of Luschka will allow the escape of cerebrospinal fluid, but they are so placed that they will not receive air". In a later paper, however, Robertson (1946) describes as "an unusual path of entry to the ventricular system observed in a case of hydrocephalus" the filling from below of ventricles which, at autopsy, showed complete obstruction by adhesions of the foramen of Magendie and of one foramen of Luschka; the remaining foramen of Luschka was enlarged and patent. We consider the excellent filling obtained in Case III to be another unusual example, and, coupled with the autopsy appearances, to be evidence of patency of the foramina of Luschka in this case.

Whilst the visualization of a high position of the transverse sinus on the X-ray films of the skull is pathognomonic of congenital atresia of the foramina of Luschka and Magendie, the absence of such a sign by no means denies the possibility of such a condition. The position of the transverse sinuses was normal in four out of the 13 cases in which it was stated. Further, the position of the transverse sinuses may not be visible in the X-ray films, especially in the skull of infants, in whom the bone is thin and the grooves cut by the sinuses are not well marked.

From an examination of the X-ray films of a number of normal skulls, it would appear that the markings of the transverse sinuses cannot be seen in the earlier months of life, and that they can be recognized only occasionally in the latter months of the first year of life, and thereafter with increasing frequency until at the age of four years the markings can be seen in most skulls.

An equally important sign in infantile hydrocephalus, at an age, as indicated above, when the markings of the transverse sinus would be most difficult to see, is the upward and anterior displacement of the posterior and inferior horns of the lateral ventricles as seen in a ventriculogram (Figure VII).

Summary.

Five cases are described in which hydrocephalus of an unusual type resulted from congenital atresia of the foramina of the fourth ventricle. Reference is made to salient features from the literature on this subject. The diagnostic significance of the finding of an elevation of the transverse sinuses and the possibilities regarding the recognition of such a sign have been discussed. The difficulty likely to be experienced in determining whether the hydrocephalus is of a communicating or non-communicating type has been emphasized, and an example is shown to support the belief that air may enter into the ventricular system *via* a patent foramen of Luschka. The value of microscopic examination of serial sections as the only reliable method of establishing the patency or otherwise of the foramen of Luschka has been demonstrated.

Acknowledgements.

We are indebted to Dr. T. Y. Nelson, under whose care these patients were admitted to hospital, for permission to make use of the clinical records. For the photography our thanks are due to the Section of Photography of the Royal Alexandra Hospital for Children, Sydney. Figures V and and X were prepared by Mr. Woodward-Smith, of the Department of Medical Artistry, University of Sydney.

References.

BENDA, C. E. (1952), "Developmental Disorders of Mentation and Cerebral Palsies", Grune & Stratton.

BUNTS, A. T., and CHAFFEE, J. S. (1944), "Agenesis of the Corpus Callosum with Possible Porencephaly; Review of the Literature and Report of a Case", *Arch. Neurol. & Psychiat.*, 51: 35.

COLEMAN, C. C., and TOLAND, C. E. (1948), "Congenital Atresia of the Foramina of Luschka and Magendie with Report of Two Cases of Surgical Cure", *J. Neurosurg.*, 5: 84.

DAVIDOFF, L. M., and DYKE, C. G. (1946), "The Normal Encephalogram", 2nd Edition, Lea & Febiger, 64.

HAMILTON, W. J., BOYD, J. D., and MOSSMAN, H. W. (1946), "Human Embryology: Prenatal Development of Form and Function", 2nd Edition, Heffer.

ROBERTSON, E. G. (1941), "Encephalography", Macmillan.

ROBERTSON, E. G. (1946), "Further Studies in Encephalography", Macmillan, 70.

RUSSELL, D. S. (1949), "Observations on the Pathology of Hydrocephalus", Medical Research Council of the Privy Council, Special Report Series Number 265.

TAGGART, J. K., and WALKER, A. E. (1942), "Congenital Atresia of the Foramina of Luschka and Magendie", *Arch. Neurol. & Psychiat.*, 48: 583.

WALKER, A. E. (1944), "A Case of Congenital Atresia of the Foramina of Luschka and Magendie", *J. Neuropath. & Exper. Neurol.*, 3: 368.

Addendum.

A sixth case has been met with since the completion of this article, and is reported by courtesy of Dr. T. Y. Nelson. A boy, aged nineteen months, appeared perfectly well save for an occipital meningocele the size of a tennis ball overlying a small hole in the occipital bone. The X-ray films of his skull showed a hiatus in the mid-line of the squamous occipital bone between the external occipital protuberance and the lambda. The sutures were not separated, and the fontanelles were closed. His head circumference was slightly large for his age (twenty and three-eighths inches, 51 centimetres). The position of the transverse sinus could not be definitely seen in the X-ray films, but there was a vague impression of a sclerotic line in the high position characteristic of a posterior fossa cyst.

The meningocele was excised by Dr. Nelson. A probe passed through the stalk could be directed freely downwards towards the base of the brain. As the stalk was above the normal position of the *tentorium cerebelli*, it was evident that a posterior fossa cyst was present. Two weeks

later a posterior decompression was performed, and a typical posterior fossa cyst was exposed. Portion of the cystic wall was removed in order to drain the cyst into the *cisterna magna*. It was not found possible to determine the patency or otherwise of the foramina of Luschka at the operation.

Post-operatively the patient did very well. On his discharge from the hospital both the decompression and meningocele wounds were flat and not bulging.

Comment.

F. D. Ingraham and H. Swan (*New England J. Med.*, 1943, 228: 559) investigating 84 encephaloceles found that 63 were in the occipital region. They state that such lesions are frequently associated with a malformation at the base of the brain, which leads to a block of the flow of cerebro-spinal fluid and to hydrocephalus.

Paul C. Bucy, in "Brennermann's Practice of Pædiatrics" (W. E. Prior Company Incorporated, 1947) states: "The occipital bone is most frequently involved in cranium bifidum. An oval defect in the occipital bone a short distance above the foramen magnum is typical."

Dandy, in Dean Lewis's "Practice of Surgery" (W. E. Prior Company Incorporated, 1932) states: "Other congenital abnormalities entirely independent of the encephalocele frequently co-exist. Hydrocephalus is very common either because of an obstruction produced by the meningocele, or because of other obstructions which are related or entirely unrelated", and in Figure 114 (page 258) he pictures an encephalocele through a hiatus in the occipital bone "communicating with the fourth ventricle and participating in the hydrocephalus caused by a block at the foramen of Magendie". It is not clear from his figure which portion of the occipital bone is involved.

Review of the skull of the patient in our Case III shows that there is a hiatus in the occipital bone, between the lambda and the external occipital protuberance, and this hiatus is covered with membrane. Histological examination shows a microscopic stalk through which the encephalocele, which had been removed at the age of seven weeks, had, in fact, communicated with the cystic fourth ventricle.

We thus have three examples, Dandy's case and our Cases III and VI, of occipital meningoceles being associated with and decompressing a posterior fossa cyst due to congenital atresia of the foramina of Luschka and Magendie. It is therefore necessary to suspect this condition when such an occipital cyst is found to emerge from a hiatus, especially if this is situated above the external occipital protuberance. Preferably the cyst should be operated upon first, since the meningocele acts as a decompression for the general hydrocephalus, and then, when a flow of cerebro-spinal fluid has been established, the meningocele can safely be removed.

Legends to Illustrations.

FIGURE V.—A lateral recess from Case I, to show a narrow channel opening into a small cyst beneath the leptomeninges. When a probe is passed through the recess a false impression is gained of an open foramen, since the probe enters the cyst and seems to move freely beneath the meninges.

FIGURE VI.—A lumbar pneumoencephalogram from Case III. The shadows of the air have been outlined. The posterior "finger-like" process is air beneath the abnormally high tentorium.

FIGURE VII.—A ventriculogram from Case III. The sclerotic marking made by the elevated transverse sinus can be seen on the inferior portion of the parietal bone. The posterior and inferior horns of the dilated lateral ventricles are displaced forwards and upwards.

FIGURE VIII.—A further ventriculogram from Case III, taken after the head has been so placed as to allow a portion of the air to pass from the lateral and third ventricles into the posterior fossa cyst. The shadows made by the air have been outlined and the triangular shadow posteriorly is due to air in the cyst.

FIGURE X.—The recess shown in Figure IX taken from one of a series of microscopic sections. The recess is closed and no cyst has developed at its mouth. Despite this, the blind recess could be easily distended by a probe passed into it, as is demonstrated in Figure IX.

FIGURE XI.—X-ray film of the skull from Case V, to show, even more clearly than in Figure VII, the high attachment of the transverse sinus.

PHÆOCHROMOCYTOMA OF THE SUPRARENAL GLAND.

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MANY more cases of phæochromocytoma are diagnosed by the pathologist *post mortem* than by the clinician in the wards. At present, if such a condition is suspected, the patient is subjected to a large number of uncomfortable, expensive and even dangerous investigations—a procedure which the clinician is unwilling to carry out unless there are fairly convincing indications for it. Lack of a simple and reliable confirmatory test is thus one reason for failure of diagnosis. Moreover, some patients have severe hypertension due to a phæochromocytoma and no history of paroxysmal episodes (Thorn, Hindle and Sandmeyer, 1944; Pratt, 1951), and in these cases the diagnosis at present is largely a matter of chance.

There is very little reference to this condition in the Australian literature, emphasized by a recent inquiry regarding the apparently negligible incidence of the tumour in this country. Only three case reports have been found since 1920 in local journals (Stokes, 1952; Lewis and Knight, 1954; Morris *et alii*, 1954), though a case of local origin has been published in Britain (King, 1931).

Ten cases were collected from four major public hospitals in Melbourne. Seven of these occurred from 1947 to 1953, and almost certainly they are the only ones which were found in these hospitals during that period. The total number of post-mortem examinations performed in these hospitals in the period was approximately 8000, so that the incidence was one in less than 1200 post-mortem examinations. This should be compared with the incidence mentioned by Blacklock, Ferguson, Mack, Shafar and Symington (1947), of only one case found in nearly 3000 consecutive post-mortem examinations in Glasgow. Also Smithwick's finding (quoted by Goldenberg, Snyder and Aranow, 1947) of an incidence of 0·5% of phæochromocytoma of the suprarenal gland in a series of 2000 patients subjected to bilateral splanchnicectomy for hypertension is worth noting here. As Smithwick also states that cases of hypertension and surgically treatable renal disease together with cases of phæochromocytoma make up less than 1% of all cases (Smithwick, 1949), it follows that, in assessing a patient with hypertension, it is at least as important to think of phæochromocytoma as a cause as to think of unilateral renal disease.

Details of four more cases were obtained, one from Launceston, Tasmania, and the others from Hobart, Tasmania, to make a total of 14. Only one case was diagnosed clinically, the remainder being unexpected post-mortem findings. In all cases sections of the tumour were available for microscopic examination, but in only two had tissue been fixed in potassium dichromate to demonstrate the chromaffin reaction.

The main features of the cases are summarized in Table I. Case VIII is reported in greater detail because of special clinical (endocrine) associations and the peculiarity of the tumour.

Clinical Record.

CASE VIII.—A man, aged forty-nine years, was first admitted to hospital in February, 1948, for investigation of pain, diminished sensation and subjective weakness in the right leg. He gave the story then of having had for three years attacks of "palpitations" lasting about five minutes. These occurred reasonably frequently and seemed to be brought on by tiredness or emotional upset.

Examination revealed the patient to be a man weighing 13 stone, with early acromegalic changes in his face and hands. His chest was barrel-shaped and the thoracic portion of his spine was kyphotic. No abnormality was noted in his heart, chest or abdomen. His blood pressure was 210 millimetres of mercury, systolic, and 135 millimetres, diastolic. Investigation of his nervous system

TABLE I.

Summary of the Fourteen Cases Studied.

Case.	Sex.	Age. (Yrs.)	Cause of Death.	Evidence of Hormonal Activity.		Associated Disease.	Tumour.		
				Clinical.	Post-Mortem.		Side.	Size.	Features.
I	F.	60	Congestive heart failure.	Paroxysms of breathlessness, chest pain and vomiting for four years. Congestive heart failure. B.P. 145/70.[1]	Slight hypertrophy of heart.	None.	R.	4 cm. diam.	Orange-brown colour. Amber pigment dissolved out in formalin.
II	M.	25	Chronic myeloid leuchæmia.	None.	None.	Chronic myeloid leuchæmia.	L.	4 mm. diam.	Small white firm nodule.
III	M.	55	Perforated duodenal ulcer.	Details unavailable.	None.	Duodenal ulcer.	R.	4 mm. diam.	Small white firm nodule.
IV	M.	71	Bronchopneumonia.	Breathlessness on exertion and at night for three weeks. Severe headaches for a "long time". B.P. 135/100.[1]	Hypertrophy of heart.	None.	L.	"Large."	Soft, friable, hæmorrhagic. Microscopically many "pseudo-rosettes" composed of elongated cells seen (Figure I).
V	F.	65	Hypertensive heart failure.	Paroxysms of palpitations, giddiness, headache and sweating for one year. Peripheral vasoconstriction noted during paroxysms. B.P. 170/100.[1]	Hypertrophy of heart. Retroperitoneal hæmorrhage and petechiæ over heart.	Serous papilliferous cystadenoma of ovary.	R.	15×12×7 cm.	Soft and hæmorrhagic with cysts filled with blood. Microscopically many multinucleated giant cells seen (Figure II).
VI	F.	37	Hypertensive heart failure.	Post-partum hæmorrhage associated with transient systolic blood pressure of 240[1] two years previously. Headaches during pregnancy. Pulmonary œdema and death following post-partum hæmorrhage.	Hypertrophy of heart. Pulmonary œdema.	None.	L.	6×5×4 cm.	Soft, pink, hæmorrhagic (Figure III). Yellow pigment dissolved out in formalin.
VII	F.	45	Congestive heart failure.	Tiredness, shortness of breath on exertion and headaches for six months. Nocturnal dyspnœa and peripheral œdema for three weeks. B.P. 250/160,[1] congestive heart failure, papilloedema.	Hypertrophy of heart, brown induration of lungs, congestive heart failure.	Serous papilliferous cystadenoma of ovary.	R.	"Hen's egg."	Firm, pale, with hæmorrhagic and gelatinous areas.
VIII[2]	M.	49	Hypertensive heart failure. Bronchopneumonia.	Hypertension and hypertensive paroxysms for at least five years.	Hypertrophy of heart.	Acromegaly and acidophile adenoma of pituitary.	R.	5 cm. diam.	"Mixed" neurocytoma and phæochromocytoma (Figure IV).
IX	F.	62	Cerebral hæmorrhage.	Attacks of weakness and inability to walk properly, often followed by vomiting, for 17 months. B.P. 240/120[1].	Hypertrophy of heart. Cerebral hæmorrhage.	None.	R.	1 cm. diam.	Greyish-pink, with central hæmorrhagic area (Figure V). Cut surface darkened on exposure to air and light green pigment dissolved out in formalin.
X	F.	28	Cerebral hæmorrhage.	Past history unavailable. Early arteriosclerotic retinopathy. Papilloedema.	Hypertrophy of heart. Arteriosclerosis. Cerebral hæmorrhage.	None.	R.	4 cm. diam.	Dark red, soft, friable.
XI	F.	33	Hypertensive heart failure. Myocardial infarction.	Headaches for twelve months. Hæmoptysis six months previously and congestive heart failure for six months. B.P. 200/140.[1]	Hypertrophy of heart.	None.	L.	"Orange".	Soft and pink. Apparent invasion of suprarenal vein. Microscopically, gross variation in nuclear size (Figure VI).
XII	M.	51	Alcoholic gastritis. Hæmatemesis and melæna.	None.	None.	Chronic alcoholism.	L.	"Golf-ball."	Dark-brown and firm. Dense fibrous stroma seen microscopically.
XIII	F.	49	—	Attacks of headache, vomiting and palpitations occasionally followed by hæmoptysis for 11 years. B.P. 240/170[1] and extremely labile. Operative removal of tumour resulted in amelioration of symptoms and a normal blood pressure.	—	None.	L.	9 cm. diam.	Brown, and contained several large cysts. Tissue darkened when placed in formalin-dichromate fixative and amber pigment dissolved out in formalin. Sevki-Schmorl stain, positive finding.
XIV	F.	35	Cerebral hæmorrhage. Hypertensive heart failure.	Lassitude, breathlessness on exertion, headaches and peripheral œdema for three years. Developed what was considered a subarachnoid hæmorrhage and died after carotid angiography	Hypertrophy of heart. Petechiæ over heart and bowel. Intraperitoneal hæmorrhage. Old and recent cerebral hæmorrhages.	Malnutrition, anæmia.	R.	4·5×3·5×2 cm.	Firm, red, hæmorrhagic (Figure VII). Amber pigment dissolved out in formalin. Sevki-Schmorl stain, positive finding.

[1] Millimetres of mercury; systolic/diastolic. [2] Full details in text.

revealed slight constriction of the spinal canal at the level of the disk between the twelfth thoracic and first lumbar vertebræ. Operative exploration was not considered advisable and he was discharged from hospital.

In October, 1950, he was referred for admission to hospital by his private doctor, who had attended him twice in one week for attacks of pulmonary œdema. Apparently his condition had remained more or less the same until about three months previously, when his attacks of "palpitations" had become more severe and more frequent. The attacks occurred nearly every morning on arising, and emotional upset also precipitated them. They subsided quickly, but were often followed by vomiting. He stated that on one occasion he had "vomited" some bright red, frothy blood. He had noticed no diminution in exercise tolerance, no headaches and no loss of weight. His libido was now absent. For three days prior to his admission to hospital he had been vomiting almost continuously and had not eaten.

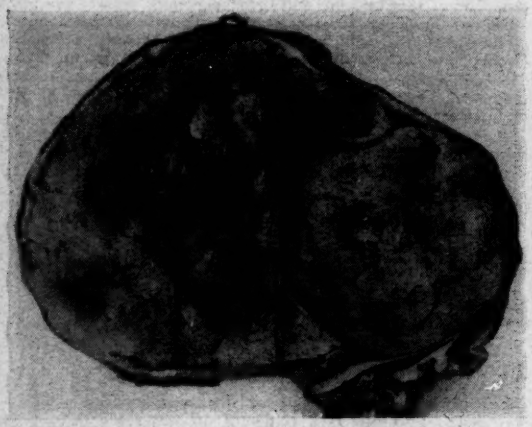

FIGURE III.
Photograph of the tumour in Case VI, which measured six by five by four centimetres.

Examination showed him to be a well-built man with moist, cold, cyanosed skin and typical acromegalic appearance—a broad head, thick nose and lips, a big tongue, broad hands with *short, thick fingers, a barrel-shaped* chest, kyphosis of the thoracic part of the spine, bowed *tibiæ and broad feet. His blood pressure was 240 milli*metres of mercury, systolic, and 170 millimetres, diastolic. His pulse was irregular, owing to multiple extrasystoles. *His heart was enlarged, and a loud apical systolic murmur,* a presystolic triple rhythm and an accentuated aortic second sound were heard. His liver edge was palpated two fingers' breadth beneath the right costal margin. His pupils were small and equal. Ocular movements were *normal and his ocular fields were normal to a simple* confrontation test. His thyroid gland was not palpably enlarged.

While he was in hospital *his systolic blood pressure* remained between 220 and 250 millimetres of mercury. In *spite of being rather dehydrated, he continued to pass* between 1·5 and 3·0 litres of urine per day, which had a specific gravity of up to 1·015 only and contained large amounts of albumin. *The blood urea content was 149* milligrammes per 100 millilitres and his haemoglobin value *was 19·5 grammes per centum.* He continued vomiting, his abdomen became distended, he became restless and irrational, and he died seven days after his admission to hospital.

At the post-mortem examination, bronchopneumonia was *found in the lower lobe of each lung.* The thyroid gland was diffusely enlarged and the parathyroids measured 1·5 by 1·0 centimetre. The heart weighed 750 grammes and there was moderately severe atheroma of the coronary arteries and the aorta. The colon in its ascending and transverse portions was grossly dilated, but no obstruction was found. The liver weighed 80 ounces and its substance was fatty and finely cirrhotic. Examination of the kidneys revealed early vascular damage. Both testes were moderately enlarged. The right suprarenal gland contained a tumour 5·0 centimetres in diameter. This was well encapsulated, and its cut surface was dark red, and mottled with areas of pale, nearly white tissue. The anterior lobe of the pituitary gland was enlarged to a diameter of 2·5 centimetres. There was erosion of the *sella turcica,* but no involvement of other adjacent structures.

Microscopic examination of the tumour revealed two types of tissue (Figure IV). One type consisted of polyhedral cells irregularly scattered in a fine connective tissue stroma. There was considerable variation in nuclear size, some deeply staining nuclei measuring 18μ by 17μ. Occasional giant cells containing two or three nuclei were

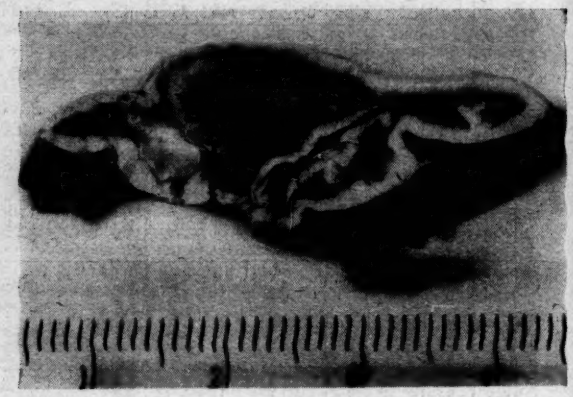

FIGURE V.
Photograph of the suprarenal gland in Case IX, showing the tumour in the medulla one centimetre in diameter.

seen, and in some areas were large numbers of cells with small, round, dark nuclei 3μ in diameter and with indefinite *outlines. Close cell-capillary relationship was seen in a* few places. The other type consisted mainly of fairly well-differentiated nervous tissue. Well-developed ganglion *cells were supported by an irregular fibrillary matrix.* This tissue was generally demarcated from the first type, but in places some of the cells appeared transitional in type and the two tissues merged imperceptibly one into the other.

By the use of Schmorl's Giemsa stain (differentiation being carried out with 0·25% acetic acid), coarse red granules closely resembling those depicted by Sevki (1934) were demonstrated in the cells of the first tissue. Differentiation with 93% alcohol as recommended by Sevki did not give this result.

Examination of sections from the pituitary gland stained by Mallory's aniline blue method showed the enlargement to be due to an acidophile adenoma.

The final diagnosis was acidophile adenoma of the pituitary gland, acromegaly, phæochromocytoma of the right suprarenal gland, hypertensive cardio-vascular disease and bronchopneumonia.

Discussion.

The clinical manifestations of phæochromocytomata have been well discussed by numerous writers (Howard and Barker, 1937; MacKeith, 1944; Blacklock *et alii*, 1947; Cahill and Aranow, 1949; Smithwick, Greer, Robertson and Wilkins, 1950).

The vast majority of these tumours are associated with hypertension. This may be sustained, paroxysmal episodes

may be superimposed on a sustained hypertension, or paroxysms may occur with a blood pressure normal at other times. In those cases in which the hypertension is sustained, there is good reason to believe that it is maintained by the pressor activity of the tumour and is not necessarily "fixed", as in many such cases the blood pressure reverts to normal after removal of the tumour (Green, 1946).

There is good evidence of hormonal activity of the tumours in 10 of the 14 cases in this series. Of the four remaining cases, in one there was some evidence of a hormonally active tumour—namely, an enlarged heart (Case IV). In Cases II and III the tumours were very small and unlikely to have any significant hormonal action, and in Case XII there was no evidence at all of activity, although the tumour was quite large. It is unfortunate that a specific stain was not used in this case.

FIGURE VII.

Photograph of the tumour in Case XIV. The tumour measured 4·5 by 3·5 by 2·0 centimetres. The main portion of the suprarenal gland is seen above and to the left of the tumour.

None of this series of tumours was regarded as malignant. In no case was metastasis found. In Case II apparent invasion of a suprarenal vein by tumour tissue was mentioned; but this was not confirmed microscopically. Brines and Jennings (1948) note that only two of the nine phæochromocytomata associated with neurofibromatosis reported to that year were associated with the "adrenal-sympathetic" syndrome, and it appears also that most of the truly malignant tumours are hormonally inactive. McGavack, Benjamin, Speer and Klotz (1942), accepting only the presence of metastasis as the criterion for malignancy, found eight cases of malignant phæochromocytoma, and none was associated with hypertension. The cases described by Spatt and Grayzel (1948) and by Blacklock *et alii* (1947) also were not associated with hypertension. However, Washington, Callahan and Edwards (1946) describe a tumour which spread along the suprarenal vein and the inferior *vena cava* and was hormonally active. Although metastases were not found and there was no other local invasion of tissues, it must be regarded as having been a malignant tumour. In two of nine cases described by Cahill (1944) the tumour had metastasized. In one the only metastasis was intracranial; from the history, the tumour was possibly hormonally active. In the other case metastases occurred widely and the history strongly suggests hormonal activity. If the presence of metastases only is accepted as evidence for malignancy, then the incidence of malignancy in phæochromocytomata is approximately 5%. This figure rises if local invasion is also accepted as evidence.

Case VIII is of outstanding interest for two reasons. Mixed tumours containing both chromaffin and ganglionic tissue are rare, and it is doubtful if the association with an acidophile tumour of the pituitary and acromegaly has previously been reported. Stimulation of the basophile cells of the pituitary gland by adrenaline does occur, and this was the probable reason for the hirsutism and sexual precocity in the case of a sixteen months old female infant with a phæochromocytoma reported by Neff, Tice, Walker and Ockerblad (1942). That the relation between the phæochromocytoma and the pituitary tumour in Case VIII may have been causal is pure conjecture; but probably the suprarenal tumour was the first to develop. When the patient was first examined the signs of acromegaly were early and minimal, and the history suggests that the phæochromocytoma had been present for three years. Of these 14 tumours, eight were in the right suprarenal gland and six were in the left suprarenal gland, nine were in female patients, and five were in male patients, and the ages of patients ranged from twenty-five to seventy-one years. These figures are in general accord with those of larger series (Blacklock *et alii*, 1947).

Before the histology of these tumours is discussed it is worth while to consider the appearance of normal suprarenal medulla. Examination of post-mortem material obtained six to nine hours after death and fixed and stained by routine methods shows irregular polyhedral cells, sometimes arranged in well-defined clumps separated by a fine connective tissue stroma (inaccurately described as an "alveolar" arrangement), but more often merely scattered diffusely through the stroma. Some cells are closely grouped around capillaries and here assume almost a cuboidal shape (Figure VIII). Often the endothelial lining of the vessel is deficient, and the cells themselves line the lumen. The nuclei are pleomorphic. Some are large, oval or irregular, and vesicular, some are very small and often pyknotic. They range from about 14μ to 3μ in their greatest diameters.

Cramer (1919 and 1928), in a study of the functional changes of the adrenal medulla of the mouse, describes the variations of cells both in the normal "resting" animal and in animals subjected to powerful stimuli, such as exposure to severe cold. In the "resting" gland two main types of cells are seen. One type contains adrenaline (or proadrenaline) granules diffusely scattered through the cytoplasm; in the other the adrenaline is largely restricted to one or more "globoid bodies", and the cytoplasm takes the special stain only very slightly. The nucleus of each type of cell is large and clear. The difference between these two types is interpreted as representing stages in functional activity. If maximal functional activity of the gland is brought about by a stimulus such as severe cold, further changes are seen. Cell outlines disappear, the cytoplasm takes the specific stain evenly, without granulation, and appears to have been "laked". The nuclei shrink and appear small, dark and sometimes elliptical. This is a description of the most extreme change, and all intermediate stages can be seen. These "laked" cells apparently do not die, but can return to their normal "resting" form.

Bennett (1941), in an excellent article discussing secretion in the adrenal medulla of the cat, noted no significant nuclear changes; but he limited his observations to the "resting" gland and to glands subjected to splanchnic nerve stimulation.

If the histology of phæochromocytomata is now considered, it can be more clearly understood. The arrangement of cells is very similar. They are usually scattered quite irregularly, although sometimes they are grouped into circumscribed clumps of five to ten cells. They are closely arranged around capillaries, forming "pseudorosettes", and sometimes are elongated rather than cuboidal when in this position (Figure 1). They may surround unlined sinusoidal spaces. Tumour cells are usually larger than those of suprarenal medulla, having, as has been mentioned by Howard and Barker (1937), a diameter of one and a half times to twice normal. The range of nuclear variation is much greater, some nuclei being 40μ in their greatest diameter. Multinucleated giant

cells are frequently seen, and occasionally groups of cells have a syncytial appearance (Figure II). Small cells with small, dark, pyknotic nuclei are often seen, sometimes in quite large numbers (Case VIII), and they have usually been regarded as immature forms, or sympathogonia, particularly in malignant tumours (*Edward, 1937*).

If the growth of a tissue is excessive and aberrant, the histological and cytological appearances in the tissue also vary, and the appearances of large cells and extremely pleomorphic nuclei in these tumours should be regarded as mere exaggerations of the normal pattern. There seems to be no good reason to regard the small cells with pyknotic nuclei as representing other than the extreme stage of functional activity. It may be that in malignant tumours they are immature cells; but in the light of the previous discussion it is thought that caution should be observed before they are so labelled. It is important, therefore, to distinguish between these cells often seen in benign tumours and the poorly staining anaplastic spindle or oval-shaped cells of truly malignant tumours, as in the case described by King (1931). Syncytial masses and giant cells may well result from the loss of normal cell outline which occurs in extreme functional activity.

Sympathetic ganglion cells are usually difficult to find in normal suprarenal medulla, but they have a common cell of origin with the "chromaffin" cells—the sympathogonia. Mixed tumours, as in Case VIII, containing both chromaffin and ganglion cells, are rare, but have been described (*Rosenthal and Willis, 1936; Blacklock et alii, 1947*), and Wahl (1914) gives a very full account of two tumours in the one patient, composed of areas of phæochromocytes, neurocytes and neuroblasts with widespread metastases from the neuroblastic portions.

The specific staining properties of the chromaffin cells have been fully discussed by a number of authors, notably Bennett (1941), Le Compte (1948), Brines and Jennings (1948) and Pearse (1953), and it is sufficient to state that the brown colour obtained in Henle's "chromaffin" reaction is a result of oxidation of adrenaline or its precursors, is not due to any chromium affinity, and is not specific for adrenaline-containing tissue except in the intensity of the reaction obtained with this tissue. Related substances in many other tissue cells give the same colour, though much less intensely.

Probably one of the easiest and most reliable stains is Sevki's modification of Schmorl's Giemsa stain (Sevki, 1934), with the use of tissue fixed in potassium dichromate. This stain can be used also for formalin-fixed tissue, as indeed it was by Sevki, when red instead of green granules are obtained. Though technically a little more difficult, the Mallory-Heidenhain azan method as described by Gomori (1946) for formalin-fixed material, giving large, well-defined, purplish-red granules in the cytoplasm, is sufficiently specific for most purposes.

With all these stains, the sooner the tissue is fixed, the greater is the chance of a satisfactory result.

It follows from this discussion that the diagnosis of these tumours presents a challenge. They are rare, but they occur sufficiently frequently to be considered in individual cases of hypertension. The pathological identity of the tumours may be established in a number of ways. Their situation and macroscopic appearance are usually characteristic. The way in which the tissue darkens on exposure to air and the diffusion of pigment into fixative are almost diagnostic. Their microscopic appearance is characteristic, and if a specific stain is applied, their identity is made certain; but failure to obtain a "chromaffin" stain may be caused by many factors and need not exclude the diagnosis. The vast majority of the tumours are benign, and the structure closely resembles normal suprarenal medulla. Most of them are simple hyperplasias.

Summary.

1. Fourteen cases of phæochromocytoma of the suprarenal gland are presented, of which only one was diagnosed clinically. The incidence of these tumours assessed from a consideration of seven of the cases was approximately one case in every 1200 post-mortem examinations.

2. An unusual case of a "mixed" phæochromocytoma and neurocytoma of the suprarenal associated with an acidophile tumour of the pituitary and acromegaly is included in the series.

3. The hormonal activity of the tumours varies. The majority give rise to paroxysmal hypertension superimposed on a normal or elevated blood pressure, some cause sustained hypertension, and some have little or no activity.

4. Most phæochromocytomata are probably not true tumours, but simple hyperplasias. Their microscopic appearances, even though the cells are usually pleomorphic and often bizarre, closely resemble those seen in normal suprarenal medulla, and most of them are benign.

Acknowledgements.

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References.

BENNETT, H. S. (1941), "Cytological Manifestations of Secretion in the Adrenal Medulla of the Cat", *Am. J. Anat.*, 69: 333.

BLACKLOCK, J. W. S., FERGUSON, J. W., MACK, W. S., SHAFAR, J., and SYMINGTON, T. (1947), "Phæochromocytoma", *Brit. J. Surg.*, 35: 179.

BRINES, O. A., and JENNINGS, E. R. (1948), "Paragangliomas: Review of Subject and Report of Five Original Cases", *Am. J. Path.*, 24: 1167.

CAHILL, G. F. (1944), "Hormonal Tumours of the Adrenal", *Surgery*, 16: 233.

CAHILL, G. F., and ARANOW, H. (1949), "Phæochromocytoma: Diagnosis and Treatment", *Ann. Int. Med.*, 31: 339.

CRAMER, W. (1919), "Observation on the Functional Activity of the Suprarenal Gland in Health and in Disease", in "Sixth Scientific Report of the Imperial Cancer Research Fund", London, Taylor and Francis.

CRAMER, W. (1928), "Fever, Heat Regulation and the Thyroid-Adrenal Apparatus", Longmans Green, London.

EDWARD, D. G. F. (1937), "Phæochromocytomata and Hypertension, with Details of Cases", *J. Path. & Bact.*, 45: 391.

GOLDENBERG, M., SNYDER, C. H., and ARANOW, H. (1947), "New Test for Hypertension due to Circulating Epinephrine", *J.A.M.A.*, 135: 971.

GOMORI, G. (1946), "Staining of Chromaffin Tissue", *Am. J. Clin. Path. (Tech. Sect.)*, 10: 115.

GREEN, D. M. (1946), "Phæochromocytoma and Chronic Hypertension", *J.A.M.A.*, 131: 1260.

HOWARD, J. E., and BARKER, W. H. (1937), "Paroxysmal Hypertension and Other Clinical Manifestations Associated with Benign Chromaffin Cell Tumours (Phæochromocytomata)", *Bull. Johns Hopkins Hosp.*, 61: 371.

KING, E. S. J. (1931), "Malignant Phæochromocytoma of the Adrenals", *J. Path. & Bact.*, 34: 447.

LE COMPTE, P. M. (1948), "Tumours of Carotid Body", *Am. J. Path.*, 24: 305.

LEWIS, R. G., and KNIGHT, L. W. (1954), "Report of a Case of Phæochromocytoma Cured by Surgical Removal", *M. J. Australia*, 1: 973.

MCGAVACK, T. H., BENJAMIN, J. W., SPEER, F. D., and KLOTZ, S. (1942), "Malignant Phæochromocytoma of the Adrenal Medulla (Paraganglioma)", *J. Clin. Endocrinol.*, 2: 332.

MACKEITH, R. (1944), "Adrenal-Sympathetic Syndrome: Chromaffin Tissue Tumour with Paroxysmal Hypertension", *Brit. Heart J.*, 6: 1.

MORRIS, J. C. H., ROBERTSON, I. F., STEVENS, F. R. T., GRANT, R. E., and STUART, A. E. (1954), "Some Current Opinions on Phæochromocytoma and an Illustrative Case Report", *M. J. Australia*, 1: 981.

NEFF, F. C., TICE, G., WALKER, G. A., and OCKERBLAD, N. (1942), "Adrenal Tumour in Female Infant with Hypertrichosis, Hypertension, Overdevelopment of External Genitalia, Obesity, but Absence of Breast Enlargement", *J. Clin. Endocrinol.*, 2: 125.

PEARSE, A. G. E. (1953), "Histochemistry: Theoretical and Applied", Churchill, London.

PRATT, W. A. (1951), "Chromaffin Cell Tumour Simulating Malignant Hypertension: Report of Case with Surgical Cure", *J. Clin. Endocrinol.*, 11: 630.

ROSENTHAL, D. B., and WILLIS, R. A. (1936), "The Association of Chromaffin Tumours with Neurofibromatosis", *J. Path. & Bact.*, 42: 599.

SEVKI, K. (1934), *"Über eine besondere Granulation der chromaffinen Markzellen der Nebenniere, ihre Beziehung zur Chromaffinität und ihr Vorkommen im Phäochromozytom", Virchows Arch. f. path. Anat.*, 294: 65.

SMITHWICK, R. H. (1949), "The Surgical Physiology of Hypertension", *S. Clin. North America*, 29: 1699.

SMITHWICK, R. H., GREER, W. E. R., ROBERTSON, C. W., and WILKINS, R. W. (1950), "Phæochromocytoma", *New England J. Med.*, 242: 252.

SPATT, S. D., and GRAYZEL, D. M. (1948), "Phæochromocytoma of the Adrenal Medulla", *Am. J. M. Sc.*, 216: 39.

STOKES, E. H. (1952), "Bilateral Phæochromocytoma", *M. J. Australia*, 1: 144.

THORN, G. W., HINDLE, J. A., and SANDMEIER, J. A. (1944), "Phæochromocytoma of the Adrenal Associated with Persistent Hypertension", *Ann. Int. Med.*, 21: 122.

WAHL, H. R. (1914), "Neuroblastomata, with a Study of a Case Illustrating the Three Types that Arise from the Sympathetic System", *J. Med. Research*, 25: 205.

WASHINGTON, R. H., CALLAHAN, W. P., and EDWARDS, E. W. (1946), "Phæochromocytoma of the Adrenal Medulla", *J. Clin. Endocrinol.*, 6: 688.

Legends to Illustrations.

FIGURE I.—Photomicrograph of the tumour in Case IV. A small blood vessel is surrounded by elongated tumour cells and no endothelial lining to the vessel can be seen. (×230.)

FIGURE II.—Photomicrograph of the tumour in Case V, showing several multinucleated giant cells. Some closely placed cells also give a syncytial appearance. (×350.)

FIGURE IV.—Photomicrograph of the tumour in Case VIII, showing the two types of tissue. Well-differentiated nervous tissue is easily recognized, and there is a localized area of chromaffin tissue above. (×75.)

FIGURE VI.—Photomicrograph of the tumour in Case XI. The large nuclei in the upper left-hand corner measured approximately 30μ × 17μ. Scattered cells with small dark nuclei which measured 3μ in diameter and gradations between the two sizes are seen. (×350.)

FIGURE VIII.—Photomicrograph of normal suprarenal medulla. Irregularly arranged polyhedral cells, pleomorphic nuclei and cells closely arranged around a small blood vessel are seen. (×350.)

Reviews.

Digital Plethysmography: Introducing a Method for Recording Simultaneously the Time Course of the Rate of Blood Flow Into and Out of the Finger Tip. By George E. Burch, M.D., F.A.C.P.; 1954. New York: Grune and Stratton, Incorporated. 8½" × 5½", pp. 142, with 83 illustrations. Price: $5.00.

ONE of the modern medical monographs published in New York is devoted to the theory and practice of plethysmography of the finger tip by the use of methods devised by Professor George E. Burch, of the Tulane University. The use of the plethysmograph is one of the oldest procedures in physiological research for recording changes in volume of a portion of the living body, at first qualitative and later quantitative. By means of an occluding cuff compressing the veins carrying blood from the region chosen, in this case the finger tip, the rate of blood flow can be calculated and the time course of such rate. A new departure here is to record the time course of the rate of blood flow out of the finger tip at the same time as the rate of inflow. An ingenious "solenoid valve" made possible the measurement of the rate of blood flow into and out of the finger tip for several pulse cycles in any preselected portion of the pulse cycle. As an illustration of the last-mentioned it is shown that in aortic regurgitation there is a high rate of flow for a brief period of time with a relatively small quantity of flow for the total pulse cycle. The many sources of error and the necessary precautions to avoid them are painstakingly examined. Thus the finger tip must be on the same level as the heart and the occluding cuff must be close to the plethysmograph, even though an artefact in the plethysmographic record is produced by its inflation. Alterations produced by varying environmental temperatures, by local warming and chilling, by peripheral and central vascular disease and by many other agents are described in detail. The whole presentation is marked by an admirable candour and absence of exaggerated claims. That finger tip plethysmography in its simpler forms could be of clinical service is evident from this work. The author might well have cited the contribution of the late Sir Leonard Hill to the physiology of circulation in rigid tissues—not that any more light would be thrown on his problems, but simply that the reader might know how the physics of circulation in such conditions had been envisaged and studied. There are a short bibliography and a good index.

Electrocardiography. By E. Grey Dimond, M.D.; 1954. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 11¾" × 9", pp. 262, with 272 illustrations. Price: £7 7s.

THIS book has two outstanding features to recommend it to the general practitioner and medical student. First, as the author assumes that for general non-specialist clinical work the direct-writing electrocardiograph is the instrument of choice, he goes to some lengths to explain the peculiarities of this instrument. Secondly, he has adopted vector principles in his study of conventional electrocardiography. He emphasizes that "to own the machine is not enough. The physician is obligated to use his machine correctly and interpret the record". As the importance of understanding the machine is all too often forgotten when a tracing is being studied, considerable detail is given to show how the characteristics of a direct-writing machine can be checked by the operator, in particular with regard to standardization, frequency characteristics and distortion of trace. A good account of the types of electrical interference which may be encountered and of methods of eliminating them is included.

A short and concise account of the principles of electrical vectors, as applied to the electrical events of the cardiac cycle, is illustrated by some spatial vector-electrocardiograms recorded from a cathode ray oscilloscope. Tracings from the conventional bipolar, unipolar and V leads are interpreted in terms of the main *P*, *QRS* and *T* vectors. This approach to the subject is in some instances very valuable and explains some of the variations seen in the relationships of the *QRS* and *T* complexes in the various leads, both within the range of normal and in abnormal conditions. An adequate discussion is given of the electrocardiogram in ventricular hypertrophy, in conduction block, in myocardial disease, in congenital heart disease, in arrhythmias and in electrolyte disturbances.

The illustrations are well reproduced and of adequate size, and a sufficient number is included to enable the book to be used as a ready reference by the relatively inexperienced interpreter.

Practical Obstetrics. By Bruce T. Mayes, M.V.O., M.B., B.S. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S., F.R.C.O.G., with a foreword by John A. Stallworthy, M.A. (Oxon.), F.R.C.S. (England), F.R.C.O.G.; *Second Edition; 1954.* Sydney: Angus and Robertson, Limited. 9" × 6", pp. 514, with 181 illustrations, 21 in colour. Price: 87s. 6d.

"PRACTICAL OBSTETRICS", now in its second edition, has been considerably enlarged. Professor Bruce Mayes has revised all the chapters of the first edition and has added 16 new chapters. The new edition is written in the same easy style as the former, and it is again emphasized throughout that the book is written for graduates, and that its purpose is to assist the young practitioner when he meets obstetric difficulties in his practice.

There is a more logical sequence of subjects in this edition. The book commences with four chapters in which pre-eclampsia, eclampsia and hypertension in pregnancy are discussed. These chapters are amongst the best in the book. Emphasis is placed on early diagnosis and treatment. Treatment is clearly set out and simple to follow. Although it is well recognized that Cæsarean section has some place in the treatment of the toxæmias of pregnancy, we do not agree with the advice that a *primipara* with toxæmia and an "unripe" cervix, in whom the membranes have been stripped and to whom "Pitocin" has been given, should be delivered by Cæsarean section if she is not in labour twenty-four hours after the manipulation.

The problems in management of the pregnant diabetic are discussed from all angles. The importance of team work in this type of case is stressed, and the good results which result from a cooperative effort are given.

The chapter on ante-partum hæmorrhage is a new one; the classification suggested is excellent and the methods of treatment are very good. The cases discussed are particularly relevant. Apart from the one case (in which a vaginal examination was carried out in the labour ward because opening the operating theatre might have influenced the decision to perform a Cæsarean section), this subject is well covered. The chapters on long labour, prolonged labour and inertia are full of helpful advice in relation to very difficult conditions. The experimental use of diathermy when the cervix is dilating slowly is not generally accepted.

Recent observation of Australian aborigines in labour showed that the oft-repeated statement that primitive people have painless labours is incorrect. "Grantley Dick Read's dream" is not a reality amongst these primitive people.

The chapter on prolapsed cord is excellent, and sets out the treatment in short and clear terms.

The sections on breech delivery and post-partum hæmorrhages are revised in this edition. The principles laid down are sound and orthodox. The illustrations are excellent. In the section on brow presentation the treatment has been oversimplified. We agree that Cæsarean section is the treatment of choice in the case in which the cervix is not fully dilated; however, vaginal delivery has a place in the obstetric management of some *multigravidæ* with full dilatation of the cervix.

The chapter on post-maturity is disappointing. The work quoted as coming from Alder (Melbourne) was carried out by Gordon Ley (Melbourne). To advocate Cæsarean section in the management of a *primigravida* who has been a sterility patient and is forty-two weeks pregnant would not be universally accepted.

Generally this new edition maintains the high standard of the first edition. In the new chapters fresh problems have been discussed from a practical point of view. Most of the deficiencies of the old edition have been made good, so that a young graduate in practice will rarely be faced with an obstetrical problem for which he cannot find the right answer in this book. It will be of great help to all general practitioners. The artist has produced many admirable illustrations, and the new publishers are to be congratulated on a fine book.

A Synopsis of Children's Diseases. By John Rendle-Short, M.A., M.B. (Cantab.), M.R.C.P., D.C.H.; 1954. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 7½" × 5", pp. 620, with seven text figures. Price: 32s. 6d.

JUST as during the last decade there have been many more popular publications written in digest or synopsis form, so also has there been an increasing number of medical textbooks published in this style. Dr. John Rendle-Short's work is one of these, and students who prefer their reading marshalled into a series of numbered lists will like this book.

The text runs into 565 pages, and as these are all in tabulated form it can be readily seen that contained therein there is a great deal of substance, presented as stated in the preface, so as to "assemble all the relevant facts tidily for easy reference and rapid revision".

The absence of illustrations will be deplored by some readers; however, if the book is used as a background to clinical studies, in place of actual photographs the student, whether he is an undergraduate or a graduate, should be able to conjure up mental pictures of actual patients, and so use the book in the true tradition of Osler.

On the therapeutic side the details given are obviously largely those of the author's own experience, and being of such a practical nature at all times they provide a most adequate guide to treatment clearly and concisely set out, which will prove popular with busy general practitioners. This book will certainly be most useful for all students and doctors who treat sick children.

Hypertension and Nephritis. By Arthur M. Fishberg, M.D.; Fifth Edition; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9¼" × 6¼", pp. 986, with 49 illustrations. Price: £6 14s. 6d.

THE reader of this book will not be misled by the author's modest statement that it is simply a book written for practitioners by a practitioner. "Fishberg" has in fact long been recognized as the most comprehensive work on the subject in the English language, and its author as a physician who has played a leading part in developing the modern concept of Bright's disease. The appearance of a new edition after a lapse of fifteen years is therefore an important and very welcome event.

Dr. Fishberg has managed somehow to survey the vast literature that has accumulated since the last edition and to add seven new chapters without materially increasing the size of the book. A comprehensive monograph can easily degenerate into an enthusiastic but one-sided exposition of the author's own views, or on the other hand, into a mere uncritical catalogue of facts and opinions, which the reviewer is tempted to dismiss, tactfully but ambiguously, as "monumental". The value of this book derives largely from the fact that the author has avoided both these common pitfalls. In discussing each topic he first briefly and clearly summarizes the relevant evidence, clinical and experimental, old and new. He then proceeds to examine the various hypotheses which have been advanced to explain the facts, and finally sums up in the light of his own unrivalled experience, wide reading and sound judgement. In doing so he displays the enviable detachment of a man who is simply aiming to get at the truth.

Each chapter is divided into numerous short sections, which are arranged in an orderly and logical way, and integration of the whole complex problem is promoted by frequent cross references. The bibliographies which follow each chapter span the literature from the earliest to the most recent references, and are refreshingly free—as is the whole book—from any trace of parochialism. In short, the new "Fishberg", even more than its forerunners, is indispensable to the clinician, the teacher and the research worker.

Hope and Help in Parkinson's Disease. By John C. Button, junior; 1953. New York: Vantage Press, Incorporated. 8½" × 5½", pp. 108. Price: $4.95.

IN an appendix to this book Dr. John C. Button has included a fascinating biography of James Parkinson. He has also reproduced in full his pamphlet, "An Essay on the Shaking Palsy", of which it might be said, as was written of another of his works, that "it at once abounds with proof of reputable talent and of an amiable disposition to benefit mankind". The appendix together with a 44-page bibliography comprises half the book. Unfortunately, the first half of the book, which is intended for the general public, is likely to give false hope and small help to the sufferer. Dr. Button, who is described as neurologist to the Elizabeth (New Jersey) Memorial Hospital and the North Jersey Osteopathic General Hospital, director of the Button Neurological Institute and member of the American College of Neuropsychiatrists, maintains that Parkinson's disease is a progressive illness only when it is not treated adequately. The reader who makes his way through a maze of padding and cheap grandiloquence will find that he recommends treatment by "injections, medicines by mouth, psychotherapy and specialized forms of physiotherapy, manipulation and massage". He states that he has given over 80,000 intravenous injections of histamine over a period of six years, and that this drug acts as a powerful tonic to the nervous system—"it seems to electrify the nerves—wake them up, make them more accurate in the transmission of impulses . . .". Of the medicines found to be beneficial he gives a list of some 170, mostly trade names, but does not indicate that he has assimilated James Parkinson's advice on the desirability of "ascertaining the actual properties of every nostrum".

Roentgenographic Technique: A Manual for Physicians, Students and Technicians. By Darmon Artelle Rhinehart, A.M., M.D., F.A.C.R.; Fourth Edition; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9¼" × 6¼", pp. 454, with 216 illustrations. Price: 91s. 6d.

THE first half of the work is taken up by elaborate descriptions of electrical set-up, with accounts of physical phenomena met with in the production of X rays and the obtaining of radiographs. This is all unnecessary in these modern days, as the radiologist pays for his physics when purchasing modern machines. He is interested only in the complicated control panel with its provisions for the correct settings to produce X-ray pictures. Expansive descriptions of how to determine penetration and to work out calibration of machines are somewhat superfluous. The author's technique is based on the thickness of the part under examination, and tables and charts of the necessary milliampere-seconds and kilovoltages are included. The actual technique of exposure is given, together with much anatomical and pathological detail; but the worker has to be continually referring back to the thickness tables for his exposure factors. This cannot be compared with other excellent treatises on technique, in which the position of the tube and the exposure factors are shown concisely for the particular part under examination. Some of the resultant films which are illustrated are not of good diagnostic value—for example, the elbow and shoulder regions. The author also passes over the value of the antero-posterior view of the flexed knee. The section on radiological examination of the mastoids is good. Altogether the work cannot be recommended for a busy radiographic department. It would be of value to students facing a higher degree examination or to the radiologist engaged in post-graduate work.

The Medical Journal of Australia

SATURDAY, NOVEMBER 6, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

GOOD DOCTORING.

DOCTORING is clearly the work of doctors, and good doctoring must be the work of good doctors. When we speak of a good doctor, we do not refer to his good behaviour or his high moral qualities, though these are obviously important characteristics of a doctor. We refer to his grasp of the subjects with which he has to deal and the way in which he uses his ability to benefit his patients. Voltaire put it very well. He wrote: "But nothing is more estimable than a physician who, having studied nature from his youth, knows the properties of the human body, the diseases which assail it, the remedies which will benefit it, exercises his art with caution, and pays equal attention to the rich and the poor." It is useful for all medical practitioners to take stock of themselves; in other words, to think occasionally of what is required of them, of the standard which they should attain, of the usefulness which they may manifest in their everyday practice. It is the function of a medical journal to draw attention to this need, and it is the function of the British Medical Association, to which the vast majority of medical practitioners in Australia belong, to take abundant care that its members are fully seized, not only with their duty in practice, but with the tendencies and temptations which beset them. We all know, for example, that since sulphonamides and antibiotics have come on the scene many an illness can be made to disappear by the exhibition of one or other, or perhaps one or two if not more, of these estimable products. The patient may recover him of his disease and return to his usual activity apparently none the worse for his adventure. He may or may not be the worse for what he has been through, because, as we now know, some of these drugs bring about a tolerance which may be far from beneficial in a future illness. Many articles have been written on the abuse of these drugs and on the need for careful diagnosis. The doctor who does everything that lies in his power to make a correct diagnosis before he prescribes a potent drug is a "good doctor"; similarly, one who does not take care as he should must perforce be a "bad doctor". The goodness or badness here, it will be agreed, constitutes a moral quality as well as a quality denoting efficiency. The British Medical Association, as cannot be stated too often, has two main objects—the advancement of the medical and allied sciences and the maintenance of the honour and dignity of the medical profession. At least one of the Branches of the Association in Australia has recently been very concerned about the standards of doctoring. It has written a circular letter to each of its members drawing their attention to matters about which complaint has been received from time to time.

The subject of good doctoring is mentioned before a reference to circular letters, because if that ideal is kept before the members of the medical profession, and if they succeed in the maintenance of their good doctor status, there will be no need for reminders from the Association, which stands, as it were, in the place of guardian angel. We may paraphrase a statement used in another sphere and say that if we seek first the advancement of the medical and allied sciences and their proper application to men and women in the community, all else that is needful will be added to us. Some of the matters which have been the subject of recent comment should be mentioned. Members of the public sometimes complain that the services of a doctor are not always available when they are needed. The occasion for such complaint can, and should, easily be remedied. When a man qualifies in medicine and undertakes practice in a certain area, he holds himself out as ready and willing to attend sick persons in that area when they require his attention. As has often been insisted in this journal, "disease knows no holiday", and any man, woman or child is likely to need the services of a doctor at any hour of the day or night. Everyone will admit that if a patient has a right to choose his doctor, the doctor has a right to choose his patient. At the same time, any doctor who is established in a certain area should be very careful before he refuses to attend a patient who calls him. He should consider all the factors which are involved in his refusal, and these factors include, of course, the public reaction to his refusal. Times may arise in cases of accident or other emergency when a doctor's moral obligation to attend a patient should overcome any hesitation that he experiences from any reason whatever. From this it follows that once a doctor has undertaken the treatment of a patient he is in honour bound to continue the attendance on such a patient. He may not say "I do not go out at nights" or "I do not attend patients on Sundays or holidays". To do this is to be recreant to his duty and to act in a manner prejudicial to the honour of his profession. In everyday language we may say that when a man undertakes to follow the profession of medicine he goes into it with his eyes open; he knows, or should know, what he is doing and he must be prepared to do it. If he goes into the profession of

medicine merely in order to earn a comfortable, or a more than comfortable, living, it would be better for him never to have turned to medicine at all. The day may well come when such a man will be thought unworthy to continue in the practice of medicine and be debarred from it. No one will suggest that the doctor should never have time free from work. "All work and no play makes Jack a dull boy" is true of the doctor as it is of every other Jack in the community. But there are proper ways and improper ways of securing this leisure. If the doctor is a member of a partnership or of a group practice, it is easy for him to let his patient know that when he is not available someone else will be on call for him, and he can then indulge in proper relaxation. If he is not a member of a group or partnership, he can and should make arrangements with other practitioners in his neighbourhood to be on duty for him at certain times; and when he does this, he must not fail to make some arrangement by which people who need his services can discover who is on duty for him. There is another point about refusal to attend patients to which reference should be made. Some who practise a specialty attend patients while they are able to get about and able to visit their consulting rooms. When such patients experience a major catastrophe, such as a coronary occlusion, they sometimes call on their specialist to attend them. The specialist sometimes answers and says that he does not visit patients in their homes, but that they should ask a local practitioner to call. The practice in cases such as this varies in different parts of the Commonwealth. In some centres the specialist would be expected, and would be willing, to make a domiciliary visit in such circumstances. In other places this would be frowned upon. If the specialist cannot attend the patient or is unwilling, for any well-grounded reason, to do so, it is he who should find a "general practitioner in the neighbourhood" to attend the patient, explaining to the general practitioner the nature of the patient's illness and just exactly what has been done for him. If this is done, the general practitioner called in an emergency will not be in such a difficulty as he will if he knows nothing whatever about the patient's illness. The patient also would at least feel that the man who has so far attended him has informed the visiting doctor what has been done and will be much more likely to have confidence in the newcomer, as well as confidence and respect for his specialist attendant.

Some patients have complained in regard to the fees that they have been charged for medical attendance. These complaints have been concerned with the total cost of an illness rather than with accounts for illnesses of shorter duration. Some patients call in a practitioner and place themselves under his care without counting the cost or having any idea of what the cost is likely to be. It would be much kinder to the patient if at the outset of what appears to be a long or costly course of treatment, the medical attendant was to discuss this with the patient. Just as it takes all sorts of people to make a world, it takes all kinds of doctors to make a medical profession. Most doctors are able to see what is happening from the patient's point of view. If they can do so, they are among the good doctors; if they cannot, they are among the bad. A doctor who insists on a patient of slender means mortgaging his home to pay for an illness or an operative procedure will not be numbered among the former.

At the present time it is very difficult for practitioners to obtain a *locum tenens* when they wish to take a holiday. Sometimes arrangements are made and a practitioner will agree to act for another for a set period. Occasionally it happens that when the time arrives, the *locum tenens* does not put in an appearance. Sometimes he abandons his post before the term of the engagement is completed. About matters of this kind it can only be stated that it is just as important to be honourable with a brother practitioner as it is to be honourable to a patient. Honour among colleagues is a feature of good doctoring; to have no thought of this is characteristic of bad. In conclusion, we may think once more of what Voltaire wrote. He insisted, first of all, that the nature of the human body should be studied; in other words, the doctor had to be perfectly familiar with the normal state. He had to study the diseases which assail the body and the remedies which would be of benefit; this has been shortly mentioned in regard to sulphonamides and antibiotics. It applies also in many other spheres. Voltaire said that the physician who was to be regarded as estimable had to exercise his art with caution; in other words, if he could do no good to his patient, he had to take care that he could do no harm. He concluded that the physician had to pay equal attention to the rich and the poor. This last point seems to be worthy of special emphasis at the present time, when some practitioners seem to demand that a full fee shall be paid for everything and everyone and, if necessary, by the State. The same practitioners talk about the freedom of the individual and demand their own freedom. It is well to remember that if too much reliance is placed on the State in the matter of the provision of remuneration for medical services, the State may some day be justified in stepping in and declaring that it will accept the whole responsibility. Than this nothing could be more calamitous.

Reference has been made to some points in connexion with good doctoring. Only if good doctoring is set up as an ideal to be pursued will the practitioner be worthy of the words which are written about him in *Ecclesiasticus* and which are read at almost every formal church service attended by doctors:

> Honour a physician with the honour due unto him for the uses which he may have of him: for the Lord hath created him. For of the most High cometh healing, and he shall receive honour of the kings. The skill of the physician shall lift up his head: and in the sight of great men he shall be in admiration.

Current Comment.

DEATHS ASSOCIATED WITH ANÆSTHESIA AND SURGERY.

A REMARKABLE survey of data relating to deaths associated with anæsthesia and surgery has been made in the United States by the anæsthesia department of the Harvard Medical School at the Massachusetts General Hospital, Boston. It is reported by H. K. Beecher and D. P. Todd.[1] The material was collected over a period of five years from ten university hospitals in various parts of the United States. The study covers analysis of nearly 600,000 anæsthetic administrations and appraisal of all deaths

[1] *Ann. Surg.*, July, 1954.

occurring in the surgical services of the ten participating hospitals during the five-year period from January 1, 1948, to December 31, 1952. Examination of the data on agents and techniques reveals that a fifth of the anæsthetic administrations in the ten hospitals were carried out by nurses—that is, twice as many as were administered by physician specialists in anæsthesia. However, the current trend is for the number given by physician specialists and resident anæsthetists to increase, and those by nurses to decrease. Inhalation anæsthesia is by far the most important single technique used, but intravenous anæsthesia has doubled in use in the period of study, while the use of the spinal technique has declined somewhat. The use of ether as a primary anæsthetic agent is steadily increasing, while the use of cyclopropane is not increasing. The closed circle filter arrangement has acquired and held an important place, but there has been a great increase in the semi-open technique. The use of the to-and-fro filter has become much less. The use of controlled respiration has increased tremendously, probably as a result of the widespread use of the muscle relaxants. Most spinal anæsthesia is carried out with tetracaine ("Pontocaine") or procaine. The use of dibucaine ("Nupercaine") has declined sharply. Heavy solutions are used more than light ones. For local or regional anæsthesia procaine is most commonly used. The use of cocaine has remained steady, but tetracaine has become much less popular. Endotracheal intubation is used principally with inhalation anæsthesia, but there has been a great increase in its use as an adjunct to intravenous anæsthesia, possibly as the result of the use of muscle relaxants. Cuffs on tubes are found to have a mortality of their own.

The information on the muscle relaxants is disturbing, or, at the very least, a cause for serious reflection. There appear to be no impressive differences in the death rate among the several muscle relaxants used, but death has occurred in association with each. Although analysis of the cause of death is difficult when combined agents are used in anæsthesia, there seems no doubt at all that the introduction of the muscle relaxants has been accompanied by a substantial increase in the anæsthesia death rate. Moreover, the evidence is against the idea that the muscle relaxants are reserved for difficult or "bad risk" cases, and so might be expected to be associated with a high death rate. The evidence also fails to provide any grounds for thinking that the experience either of the anæsthetist or of the institution provides protection from disaster with muscle relaxants. An important observation is that, despite artificial respiration of a generally effective type, many patients to whom muscle relaxants are given die of circulatory collapse. Another point of interest is that the combination of a muscle relaxant and thiopental is not as dangerous as the combination with ether or cyclopropane. Passing sober judgement on these findings, Beecher and Todd state that they do not believe that the muscle relaxants should be banned, but that they should be studied further. Advances, apparently, are being made. The data presented strongly suggest that great caution is indicated in the use of the muscle relaxants, that the agents available at present should be considered as on trial, that they should be employed only when a clear advantage is to be gained by their use and that they should not be employed for trivial purposes or as a corrective for generally inadequate anæsthesia.

The study of the specific data on death rates of surgical patients reveals that, in terms of the deaths in these ten hospitals scattered throughout the United States, a patient has a chance of one in 75 of dying from one cause or another. He has a chance of one in 95 of dying from the disease which brought him to hospital, and the over-all anæsthesia death rate is one in 1560; this figure represents cases in which it was agreed that anæsthesia was important in the death, although not always the primary cause. Men appear to have a disproportionately high anæsthesia death rate. The cause for this is not quite clear, but it may be related to the tendency of the breadwinners of the family to postpone going into hospital till, in some cases, their physical condition is not as good as it might have been. Perhaps the incidence of serious heart and circulatory disease in men makes them less able to tolerate anæsthesia than women. Examination of the data on age reveals the important finding that anæsthesia deaths are disproportionately great in the first decade of life and in the later decades.

Summing up certain general observations that emerge from this study, Beecher and Todd state that great changes in the use of anæsthetic agents and techniques occurred within the five years of the study. This suggests that the practice of anæsthesia is as yet far from achieving stability. They point out also that the inaccuracies of clinical impression in this field, as opposed to the evidence of carefully recorded fact, are illustrated by five examples: the place of ether anæsthesia, the place of cyclopropane anæsthesia, hazards in the use of muscle relaxants, the usefulness of the nurse-anæsthetist, and the death rate attributable to anæsthesia. A broad conclusion from the study is that death from anæsthesia is of sufficient magnitude to constitute a public health problem. Anæsthesia kills several times as many citizens each year out of the total population of the United States as does poliomyelitis. As Beecher and Todd point out, consideration of the millions of dollars rightly spent in attacking poliomyelitis and the next-to-nothing, comparatively, spent in anæsthesia research makes clear an urgent need. There are plenty of avenues opened up in this study along which research might profitably be conducted. The study sets out clearly to show only what the situation is, not what it should be. The latter problem merits careful attention.

TOXIC HAZARDS IN HOBBIES.

WE are used enough to toxic hazards associated with industry and are generally alert to their occurrence, but quite illogically many people will feel a jolt on reading such a statement as the following, made by Marion Gleason:[1] "A health hazard of which physicians are not always aware lies hidden in the toxic chemicals used by hobbyists in such apparently harmless pursuits as stamp collecting, gardening, craft work, insect collecting, outboard-motor boating and photography." It is, of course, not really a surprising statement, but it provides a useful stimulus to the medical practitioner to make himself better informed on the subject. Gleason's article touches on some of the most important points. The prize for the most prevalent toxic hazard in hobbies goes to carbon tetrachloride. As Gleason puts it, "there is scarcely a leisure-time activity that does not provide opportunity for the use of this efficient, non-inflammable, but extremely toxic liquid". It is used as a quick killer of butterflies and beetles, instead of cyanide, ether and chloroform, as a stamp cleaner and in water-mark detection dishes, in preference to the inflammable benzine, as a cleaning fluid and solvent in craft work, as a film cleaner in photography, as a component of insecticides in the garden, and so on. In furniture making, upholstery work and the like, which are often hobbies as well as trades, carbon tetrachloride appears in waxes and polishes as well as in rug and upholstery cleaners, and "its treacherous fumes take their toll in victims chronically or episodically ill with damaged livers or kidneys, or even killed by lethal exposures". In industry its use is controlled and its safety concentrations under various circumstances are carefully determined, but the blissfully ignorant stamp collector, for example, often works much beyond the safety limit. Apparently a good rule with carbon tetrachloride, or with any of the halogenated hydrocarbons, is: "If you can smell the odour, the exposure is dangerous."

Of other poisons used in hobbies there is quite a variety, and they are listed in a useful table in this article. A few are given special mention. Potassium cyanide, which is treated with universal respect when it appears openly, may lurk in silver and metal polishes. Carbon monoxide can

[1] *Practitioner*, June, 1954.

threaten the motorist, not only in a closed garage, but in traffic jams and during running with closed windows. It also requires caution from devotees of outboard motors. Turpentine, used by the amateur painter and craftsman, is readily absorbed by inhalation and through the skin. Giddiness and inebriation may result, with nephritis on prolonged exposure. Poisonous substances in great variety are used as pest and weed killers—many of them much more harmful than the well-publicized DDT. Nicotine, like some other agents including carbon monoxide, can cause mild effects which make the person concerned more susceptible to accidents. Apparently toxic amblyopia, due to cigarette smoking, can be made suddenly acute by ingestion of alcohol and so play a part in accident production. This is only one of the hazards incurred by those who include alcohol and nicotine among their hobbies.

While the obvious solution to these problems of toxicity is to ban the poisons concerned from their present uses, this cannot be readily justified in all cases. Used sensibly they are as safe as many other things in our rather hazardous modern world, and there may not be a satisfactory substitute. It is important, however, that doctors be aware of the possible sources of unusual symptoms, and that users of poisonous substances understand what they are doing. Not the least effective measure would be encouragement as a hobby of the much-neglected practice of reading the labels on bottles.

BOWEL CONTENT DEODORIZATION BY CHLOROPHYLLINS.

WHATEVER may be the truth about the absorption of odours by water-soluble chlorophyll derivatives or "chlorophyllins", there is considerable evidence that these substances are active in bowel deodorization, so that subjects with colostomy who ingest them lose their offensive smell. Chlorophyllin deodorization has been variously ascribed to (i) catalytic oxidation or reduction of volatile odoriferous materials, (ii) stoichio-chemical combination and fixation of these materials and (iii) an antibacterial effect wherein the porphyrin is presumed to act like an antibiotic and to prevent production of odorous material by bacteria. Oscar Coren and R. D. Barnard[1] have studied the effects of a cupri-complex of chlorophyllins called "Chloresium" on certain intestinal bacteria known to produce offensive substances by putrefaction. Patients ingesting "Chloresium" frequently develop a Gram-positive saccharolytic intestinal flora preponderance. Cultures of four hydrosulphide-producing, non-indol-producing enteric organisms—*Proteus vulgaris*, *Salmonella dysenteriæ*, *S. typhi* and *S. schottmülleri*—were grown on S.I.M. agar and triple sugar-iron agar made to contain 0·0001%, 0·001% and 0·01% of "Chloresium". There was a sharp diminution of hydrogen sulphide production by all the organisms after the second transplant to these chlorophyllin-containing media with the higher initial concentrations of the porphyrin (0·001% and 0·01%). Chlorophyllin was decolorized by the growth of these organisms. No other effect on bacterial growth was noted, but there was an entire absence of any detectable odour in any culture containing chlorophyllin, even in those instances in which hydrogen sulphide production could be detected by the ferric indicator incorporated in the medium used (black colour due to ferric sulphide formation), as well as in those instances in which the pigment had been decolorized. Cultures grown on media not containing chlorophyllin had a fæcal odour most obnoxious in the case of *Proteus vulgaris*. Cultures of *Endamœba histolytica* did not develop a putrescent odour when chlorophyllin was present in the media. The presence of the pigment evidently produced some metabolic change in the bacteria. Just how this could be produced is not known, but the action resembles that of an antibiotic. Organisms which have become resistant to an antibiotic have altered metabolism. On the basis of these experiments, about 10 milligrammes of dispersed chlorophyllin in the contents of the lower part of the bowel should be sufficient to deodorize the contents; but five to ten times that amount must be ingested daily by a patient on an ordinary diet to effect deodorization, owing no doubt to destruction in and absorption from the higher part of the bowel.

[1] *New York State J. Med.*, August, 1954.

CURRENT MEDICAL RESEARCH IN AUSTRALIA.

AN encouraging impression of the nature and scope of medical research being carried out in Australia is conveyed by the report recently issued by the National Health and Medical Research Council of work done under the *Medical Research Endowment Act* during the year 1953. The amount appropriated by Parliament for medical research in the year was £129,500. In fact, a further £11,500 was spent, but this was covered by a balance in the endowment fund at the beginning of the year. Of the many research projects being carried out in approved research institutions, the report lists a limited number as being of more popular and general interest. Quite an extensive work is being carried out at microbiological typing centres. At the University of Adelaide the Salmonella Typing Centre, using type sera and phage, during the year investigated 425 cultures sent from various parts of Australia and New Guinea. The most abundant type was *Salmonella typhi-murium* followed by *S. seftenberg*, the latter being isolated entirely from contaminated coconut. A new strain *S. hvittingfoss*, which was responsible for a small outbreak of food poisoning in Brisbane, was isolated for the first time in Australia. At the Fairfax Institute of Pathology in Sydney, staphylococci responsible for infections are typed by phage. The high incidence of penicillin-resistant strains in hospital in-patients still continues, and 57 strains, as compared with 35 strains isolated in 1952, were found to be resistant to all five antibiotics, namely, penicillin, streptomycin, "Aureomycin", chloramphenicol and "Terramycin". The report notes that virus reference laboratories have been established in Brisbane, Melbourne and Adelaide. In Melbourne, the technique of complement fixation for the detection of antibody of Murray Valley encephalitis has been established, and during the summer of 1952-1953 specimens of serum were examined from 60 clinical cases of encephalitis, but none were due to the Murray Valley encephalitis virus. Specimens of serum from 243 residents of Broken Hill were tested for Murray Valley encephalitis antibody, and a significant difference was observed between older residents and those under the age of thirty-five years. This finding is considered to point strongly to the occurrence of immunization against the Murray Valley encephalitis virus at the time of the "X" disease epidemic of 1917 and 1918. At the Adelaide unit, serological evidence was obtained which suggests that endemic areas of Murray Valley encephalitis exist in northern Australia. Also in Adelaide, tissue culture passage of the poliomyelitis virus is working satisfactorily. The unit there is undertaking the periodic isolation and typing of strains as they occur in Australia. A virus which was isolated from a subject of meningitis at Port Augusta has been found to have some of the characteristics of psittacosis, and its relationship to this group is being investigated. Work carried out in both Melbourne and Adelaide has indicated that the awareness of psittacosis infection is low in Australia. Although recent serological surveys in Adelaide have shown the presence of antibodies in 25% of the population, it is assumed that the disease must have been of subclinical degree in most cases. In North Queensland, work has been concentrated on rickettsial infections, and some strains of scrub typhus rickettsiæ have been isolated.

In the field of biochemistry, investigations have been of a fundamental nature, and valuable data on the mechanism of metabolism in its many aspects are being collected which will, no doubt, in the future play a part in the understanding and elucidation of some of our problems.

A variety of clinical research projects have been carried on. Particular attention is drawn to the work being carried

out at the Royal Melbourne Hospital on the treatment of duodenal ulcer. Following a technique developed during the previous year, surgeons at that hospital have now treated 50 patients by combined deep X-ray therapy with excision of the ulcer and the neighbouring antrum. The results so far have been encouraging, and one of the surgeons concerned has been invited to lecture on the technique in London and the United States. Also in Melbourne, liver biopsy has been used as an additional method of investigation of patients suffering from pyrexia of unknown origin or from obscure radiological lesions in the lungs when other methods have failed to establish a diagnosis. This method is found to be of use in the diagnosis of tuberculosis or sarcoidosis.

Amongst the developments in physiology and pharmacology, a new technique has been devised which, it is considered, should be of great value in endocrinological and physiological research. Small plastic needles can be planted in any gland or muscle or a ventricle of the brain and then attached to an injecting mechanism, the whole being buried in the experimental animal. By this method, the injector can pump continuously and constantly for periods up to three weeks. Work has also been carried out on the development of a new barbiturate antagonist.

In the study of the influenza virus, an important finding has been obtained in virus genetics that virulence is determined, in many instances at least, by the "dosage" of a single genetic unit. When an avirulent strain is crossed with a virulent one, there follows a series of viruses with greater virulence. The implications of this concept for the process of adaptation to new hosts are now being studied. Influenza typing of the extensive epidemic which spread through Melbourne in August and September, 1953, showed it to be due to a type B virus and closely related to strains isolated in Melbourne during 1948 and 1951. Two strains of influenza isolated in Queensland were identified as type A and were closely related to strains current in Melbourne in 1950 and 1951. The chronological change in the antigenic pattern of the type A or B influenza virus is described as a matter which needs continuing observation. The change has been progressive and has never been observed to be "backward".

These are only examples of the work being carried out under the terms of the Act, but they are sufficient to show a significant amount of activity in Australian research institutions.

HYPERTENSION: HUMORAL AND NEUROGENIC FACTORS.

DESPITE a vast amount of clinical and laboratory investigation hypertension remains a subject of mystery. For obvious reasons it remains also a subject of great interest. The latest Ciba Foundation Symposium[1] deals in the main with the functional aspects of hypertension. Thirty-three experts from Europe and America met to discuss papers on many approaches to the subject, and the discussion after each paper was free and often pointed and is not the least valuable part of the volume. One thing seems to stand out, and that is that there were almost as many views on hypertension, its causation and effects, as there were speakers. There were forty papers, which covered a wide field. Views on the relation of the kidneys to hypertension were very varied. At least three substances obtained from the kidneys were considered by some to have causal relationship to hypertension and its effects. On the other hand Grollman brought forward evidence to indicate that hypertension is a result of a defect in an incretory function of the kidney.

It seems clear from the papers and discussions that more than one mechanism can cause hypertension. What relation essential hypertension, as it occurs in man, has to the types of hypertension produced in animals by the various experimental methods described does not yet seem obvious. The papers are for the most part of high quality and the discussions very stimulating. There is little that would assist a physician in the treatment of hypertension but much to interest him.

[1] "Ciba Foundation Symposium on Hypertension: Humoral and Neurogenic Factors." Editors for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8" x 5½", pp. 308, with 73 illustrations. Price: 30s.

HIGH CALCIUM INTAKE IN PREGNANCY.

CALCIUM plays an important part in uterine activity during the various stages of labour. Post-partum uterine contractions do not occur in the dog and in the rabbit when available calcium is sufficiently reduced. In the latter part of pregnancy there is a fall in the serum calcium level, whilst the requirements of calcium and phosphorus then are almost double those of non-pregnant women. Taking these facts into consideration, S. Gold[1] has given an increased supplement of calcium in the last month of pregnancy. There was no modification of diet and the patients continued to take vitamin and mineral tablets. Sixty public hospital patients were given calcium gluconate, a ten-grain tablet three times a day an hour or so before meals. To 40 private patients the extra calcium was given as purified bone meal in a proprietary preparation containing vitamins. The progress and character of labour in all the patients taking increased calcium compared favourably with that in control cases. Once started labour progressed steadily, and there was good uterine relaxation between contractions. There was hardly any noise and no hysteria or panic right through the first stage of labour. The duration was much shorter and labour more efficient than in the control cases in the second stage of labour. The third stage and post-partum course were uneventful. The increased intake of calcium did not seem to have any appreciable effect on false labour pains, and no deleterious effects were encountered.

PETRI DISHES.

THE STANDARDS ASSOCIATION OF AUSTRALIA announces the issue, for public critical review, of a proposal to endorse B.S. 611:1952, Petri Dishes, as an Australian standard (Doc. 276). The endorsement is subject to amendment, primarily the omission of a test for quality of glass. The Association's Committee on Laboratory Glassware has been concerned for some time with the question of quality of glassware, particularly for Petri dishes. The autoclave test of the British standard was considered to be too lengthy and involved, and alternatives such as drop tests have been studied. After a series of tests, the committee has accepted the view that the visual examination for clear and smooth surfaces is most important and that there is as yet no satisfactory test worthy of incorporation in the standard. The current British standard takes into account the increased importance of manufacture by pressing instead of blowing into the mould. This requires a thicker wall than is desirable in a blown dish, and separate sets of limits have therefore been included for the two types. Problems of frosting and correct fitting and the high breakage rate were also matters of concern to the committee, which feels, however, that the British standard, as amended, covers all that can be included at this stage. The committee will welcome constructive comment on B.S. 611 and the proposed Australian amendments thereto. Copies of B.S. 611:1952 can be seen at, or purchased from, the headquarters of the Association, Science House, Gloucester Street, Sydney, or from branch offices in other capital cities and at Newcastle. Copies of Doc. 276 can also be obtained at these offices. Any comment should be forwarded to reach the Association not later than December 22, 1954.

[1] *Canad. M. A. J.*, August, 1954.

Abstracts from Medical Literature.

OTO-RHINO-LARNGOLOGY.

Keratosis of the Larynx.

F. J. PUTNEY AND J. J. O'KEEFE (*Ann., Otol., Rhinol. & Laryngol.*, June, 1953) state that there are cases of laryngeal keratosis which have nothing in common with carcinoma. In other instances the condition leads to carcinoma, and in a third group keratosis develops as a sign of carcinoma. Since it is impossible to foretell the destiny of any such areas, the elimination of all should always be aimed at. In simple keratinization great improvement will follow within two or three weeks after removal of all forms of irritation and abuse of the larynx. If definite clinical evidence of healing has not appeared after this time, the lesion must be regarded with suspicion. Persistent or recurrent keratosis requires repeated histological study, including that of the subepithelial layer, to detect malignant change, which is the usual outcome. Treatment with irradiation may produce disappearance of the keratotic lesion while it is still in the pre-malignant stage. Distinction between simple keratinization and carcinoma-in-situ in the larynx can be recognized by the pathologist, but the lesion should be evaluated by the laryngologist and the method of treatment dictated by his clinical experience.

Problems in Vasomotor Rhinitis.

H. P. HARKINS (*Arch. Otolaryngol.*, May, 1954) states that the term vasomotor rhinitis is a confusing one. There are the allergic cases in which positive skin test reactions are obtained, or those in which the reactions occur on contact with specific substances. In this discussion consideration is given to the very large group in which no positive skin reactions occur. The most important single finding in the diagnosis of these cases is the presence of eosinophile cells on examination of the nasal secretions. Such examination should be carried out routinely in all suspected cases. Intradermal tests with histamine diphosphate 1:100 to 1:1000 will frequently give a marked reaction. If polypi are found, they should be removed; obstructive septal deflections and turbinal enlargements should be corrected. Electrocoagulation of engorged turbinal tissue is helpful. Probably the most gratifying recent advances in the therapy of vasomotor rhinitis have been in the field of histamine desensitization and non-specific antigen therapy. Many of the patients are greatly helped by house-dust injections, even though skin test results are negative. The injections are given twice weekly, commencing with 0·1 millilitre of 1:100,000 dilution. The dose is increased by 0·1 to 0·2 millilitre each visit until one millilitre is reached, and then the same schedule is repeated with the 1:10,000 dilution. The programme is again repeated with 1:1000, 1:100 and 1:10 dilutions until definite lasting improvement is noted. Histamine diphosphate may be used in several dosage schemes. Subcutaneous injections of increasing doses of the standard 2·75 milligrammes in five millilitres of diluent may be given, 0·1 millilitre of this solution being the usual initial dose. The doses are increased by 0·1 millilitre each until relief is obtained. The injections are repeated twice a week, but the dose is not increased once relief is obtained. In the optimum dosage method the patient receives injections of histamine diphosphate varying in concentration from 1:100,000 to 1:10,000,000. The strength of the dose given depends on a careful intradermal titration in which 0·5 to 0·1 millilitre of each strength is introduced just below the outer layer of the skin in areas about one inch apart. The dilution which produces a weal between 17 and 19 millimetres in diameter is the one selected for therapy. An injection of 0·1 millilitre of this strength is given subcutaneously, and the amount is increased by 0·1 millilitre at each subsequent injection until relief is obtained. The dose is then no further altered in amount and is repeated when there is cessation of comfort, which may be at the end of five or seven days or may not be necessary for three or four weeks. Antihistaminic drugs and vasoconstricting sprays may be employed during the wait for full therapeutic effects from the injections.

Spontaneous Hæmorrhage, with Special Reference to Epistaxis.

P. JACOBSON (*Arch. Otolaryngol.*, May, 1954) states that the character and significance of epistaxis have never been understood. It is suggested that spontaneous hæmorrhage is a clinical entity in itself and that epistaxis is the commonest form of this disease. Over 300 patients with nose bleeding have been treated by the author with intravenous administration of œstrogens. The red cell, white cell and platelet counts have been found to be uninformative. The coagulability of the blood plays no part, since in all instances it was normal. Blood pressure does not appear to be of significance. Eight cases of epistaxis which was controlled with large doses of œstrogens are discussed. The patients would have required extensive and perhaps repeated packings and cauterizations to control the severe hæmorrhage without the œstrogen therapy. Usually 20 milligrammes were injected intravenously, and hæmorrhage ceased within twenty or thirty minutes. In some instances there was a recurrence a short time later, and again cessation resulted from a similar injection. In three other cases in pregnant women control was effected with oral and intramuscular administration of œstrogen only. In a fourth case bleeding was from the lower part of the throat of a boy of fifteen years. Hæmorrhage ceased twenty minutes after intramuscular injection of œstrogen. Hæmorrhages may occur spontaneously from many other parts. A broad experience with œstrogens in all of these conditions warrants the opinion that they are not dissimilar. Specific lesions such as ulcers, blood dyscrasias, trauma, infection and tumours may provide a source for bleeding, but may not be the true cause of hæmorrhage. The theory of fluctuating hormones may explain these hæmorrhages, and what is considered to be many diseases may be a single disease with a multitude of manifestations. Favourable response to œstrogen therapy has been induced too many times for the event to be discussed as a fortuitous circumstance. It is considered that episodes of spontaneous hæmorrhage, including epistaxis, are not isolated instances in themselves, but are the manifestations of a systemic disease of which bleeding is the principal sign. They arise out of the reduction of circulating œstrogens, and the deficiency excites the formation of a tissue toxin that promotes vascular catabolism, especially of the veins. The cause of the decline is unknown, but it may be due to emotional complexes. Other hormones may be involved. A conjugated œstrogenic substance was used in treatment of the series of patients.

Treatment of Ozæna and Rhinopharyngitis Chronica Sicca with Vitamin A.

A. E. STRAUDBYGARD (*Arch. Otolarngol., April, 1954*) states that for years he treated patients suffering from ozæna, rhinitis and *pharyngitis sicca* with vitamin A in small doses taken over prolonged periods. When the production of synthetic vitamin A was effected and larger doses were thus made available, results were obtained in months which previously had taken years to produce. Of a series of 12 cases of ozæna treated in Denmark the mucosa returned to normal in nine. In three there was failure to respond. Relapse tended to take place on withdrawal of the vitamin A concentrates, but the normal condition was reestablished upon repeated administration. Daily maintenance doses of 100,000 to 200,000 international units of vitamin A seem to be required. At the request of the author the treatment was used by colleagues in Berlin. Twenty-three patients with *rhinitis atrophicans* and ozæna were given 300,000 international units of vitamin A ("Arovit"-Hoffmann La Roche) intramuscularly twice weekly and 150,000 to 300,000 international units by mouth on the remaining days. During the second or third week patients reported spontaneous loosening of the crusts and relief from headaches. Crust formation and fœtor disappeared, and the mucosa presented a fresher moist appearance. *Pharyngitis sicca* proved most resistant. Symptoms of ozæna reappeared after discontinuance of "Arovit" therapy for five weeks. In five cases of the 23 the condition proved refractory to the treatment. No untoward symptoms were produced from the high vitamin A intake. The experimental findings of Fell and Mellanby that in embryonic chick ectoderm keratinization was suppressed and differentiation into mucus-secreting, often ciliated epithelium was caused by large amounts of vitamin A, lend support to the author's views.

Repair of Tympanic Membrane Perforations with Human Amniotic Membrane.

W. J. SCHRIMPF (*Ann. Otol., Rhinol. & Laryngol.*, March, 1954) states that the tympanic membrane must be intact in order to have a functionally perfect acoustic organ. Any perforation or loss of substance must result in some impairment of normal function. Various materials have been employed either to close over a tympanic perforation temporarily or to act as a splint under which healing might be expected to take place. Amongst these cigarette paper, waxed paper, "Cellophane", gold leaf, and prepared sheep's mesentery have been employed. Several substances have been employed in an endeavour to freshen the margins of the perforation and to stimulate reparative processes; silver nitrate, trichloracetic acid and "Euthymol" have each

been of some effect. Human amniotic membrane is thin, transparent, tough and elastic. In addition, the mesodermal chorionic side of the amnion adheres quite tenaciously to almost anything with which it comes into contact. The author prepares human amniotic membrane in the following manner. Several large clear pieces are cut free from the placenta and are placed in sterile sealed brown glass jars containing Ringer's solution with "Merthiolate" one part per thousand of solution. It has been found to be sterile after twenty-four hours and has so remained for as long as four weeks. In all the following steps the membrane must be handled aseptically. The membranes are first stretched out on a sterile towel to absorb most of the solution. Next the chorion is separated from the amnion by stripping with sterile forceps and scraping with a dull all-metal table knife. The side to which the chorion was adherent must be noted, as this is the adherent mesodermic side, which will be applied against the tympanic membrane. The amnion is then cut into circular pieces such as will fit into a selected Petri dish, which is fitted with a piece of corrugated cardboard thoroughly impregnated with paraffin and which has all been sterilized in an autoclave. A piece of the damp amniotic membrane is next stretched with the fœtal side downwards over the paraffined cardboard and fixed with pins. The cardboard and stretched membrane, together with a small waxed container of calcium chloride to absorb moisture, are then placed within the Petri dish and covered over. This is allowed to dry for forty-eight hours or longer. Transfer may be made to another similarly treated Petri dish if drying is incomplete or should drops of moisture form inside. After the membrane is thoroughly dried, it presents a thin transparent appearance somewhat like "Cellophane" and is easily handled. With a metal punch pieces of various sizes are cut out, and these are stored ectodermal side up on a square of sterile waxed paper, which is then folded over as a pharmacist puts up powders. The packages are stored in a sterile Petri dish until needed. To apply the pieces to a perforation, the margins are first touched with 50% trichloracetic acid solution, and then a piece of amniotic membrane is applied mesodermal surface downwards against the perforation to cover it completely. Closure was successful in 48 of 53 cases treated. Improved hearing was usually obtained, the average gain in all frequencies in the air conduction audiogram being 18 decibels.

Sympathetic Surgery in Ménière's Disease, Tinnitus Aurium and Nerve Deafness.

L. F. JOHNSON (*Arch. Otolaryngol.*, April, 1954), in the light of the incomplete understanding of the pathogenesis of the Ménière syndrome, suggests the indefinite term neuro-vascular dysfunction as a broad explanation. He states that from some experimental observations it may be deduced that sympathetic stimulation may reduce the blood supply to the labyrinth. The control centre of the autonomic system rests in the hypothalamic region of the brain. Afferent impulses to this centre from other regions and from the emotions may disrupt autonomic equilibrium with resulting vascular spasm. Dilatation of the blood vessels of the membrana tympani has been observed following stellate ganglion block. Passe also observed during a fenestration operation dilatation of the blood vessels of the membranous labyrinth when the stellate ganglion was blocked. Stellate block in the course of Ménière's disease can effect a rapid and dramatic relief from symptoms. It is suggested that acute Ménière's disease or acute deafness should be handled as an acute surgical emergency. There may be very little time to effect the desired change in the endolymphatic system if permanent deafness is to be averted. It is proposed that as soon as severe Ménière's disease is diagnosed the stellate ganglion should be blocked with procaine hydrochloride. In two cases met with early in acute attacks of Ménière's syndrome such stellate ganglion block produced relief from symptoms in three to four minutes coincidental with development of Horner's syndrome. For patients with recurrent symptoms a dorsal sympathectomy is advised for a more permanent effect. In this the second, third and fourth sympathetic ganglia are decentralized. Tinnitus may often be relieved by these procedures, the effect of procaine block being tried first. A similar procedure may reasonably be tried in some cases of nerve deafness. Improved hearing of the low frequencies has been observed in certain cases.

OPHTHALMOLOGY.

Ocular Manifestation of Disturbances in Calcium Metabolism.

FRANK B. WALSH AND ROBERT G. MURRAY (*Am. J. Ophth.*, December, 1953) review the physiology of calcium metabolism and the biochemistry of those conditions causing hypercalcæmia and hypocalcæmia. They state that the ocular signs of hypercalcæmia may be calcification of the cornea and conjunctiva in the absence of any ocular inflammation. This metastatic calcification may be an early and in fact the only sign of the disorder. The authors divide hypoparathyroidism into three types: idiopathic or primary hypoparathyroidism, pseudohypoparathyroidism, and acquired or post-operative hypoparathyroidism. They state that the important ocular signs in idiopathic hypoparathyroidism are conjunctivitis, keratitis, blepharospasm, cataracts, papillœdema and calcification in the basal ganglia. The presence of phlyctenular conjunctivitis, persistent conjunctivitis, keratitis and blepharospasm should always call for an estimation of serum calcium and phosphorus contents. Cataracts develop in the majority of patients with hypoparathyroidism, and in most cases slit-lamp evidence consists of dot-like opacities situated rather well away from the capsule. In some cases lens extraction becomes necessary. Papillœdema has been reported in many cases and is characterized by signs of parathyroid deficiency which have been present for years before the onset of severe symptoms; pronounced hypocalcæmia is always present when papillœdema is diagnosed, the papillœdema is of low grade, and spinal fluid pressure is not unduly high. The spinal fluid has normal calcium and phosphorus contents. Papillœdema in a patient with convulsions or with a history of convulsions should always suggest hypoparathyroidism, just as do convulsions without papillœdema. In addition, in cases of unexplained papillœdema the serum calcium content should be estimated, since latent tetany is easily overlooked. Radiologically visible calcifications in the basal ganglia have been present in several cases of hypoparathyroidism. Referring to local disturbances in calcium metabolism, the authors discuss calcification in the cornea and choroid, intracranial calcification, that associated with parasites, arterial calcifications and the calcifications associated with angioid streaks and pseudoxanthoma elasticum.

Air Injection in the Treatment of Traumatic Hyphæma.

JOHN W. WILSON *et alii* (*Am. J. Ophth.*, March, 1954) present their method of preventing the disastrous secondary hæmorrhage which often occurs after a slight traumatic hyphæma. They injected air into the anterior chamber in 27 cases, and secondary hæmorrhage occurred in only two. Their routine treatment in all cases of traumatic hyphæma is complete bed rest for four or five days. Air is injected into the anterior chamber, and the injured eye only is occluded. No local medication is used, except atropine after the fifth day if there is evidence of iritis. Sedatives are administered. The technique of air injection and removal of blood clot from the anterior chamber is outlined.

Hydrocortisone in Ophthalmology.

DAN M. GORDON (*Am. J. Ophth.*, April, 1954) reviews his experience with hydrocortisone used topically, subconjunctivally and orally. He found that topical applications were more effective clinically than cortisone. In some cases hydrocortisone succeeded in patients who had no response to cortisone, and in this respect cases are quoted of vernal catarrh which was controlled only on exhibition of hydrocortisone. The author also found that, as with cortisone, diseases of the anterior segment respond more effectively than diseases of the posterior segment. For posterior segment inflammations he recommends an initial course of systemic therapy followed by subconjunctival and topical therapy as the disease responds.

The Relationship of Retrobulbar Neuritis to Multiple Sclerosis.

ROBERT G. TAUB AND L. WILBUR RUCKER (*Am. J. Ophth.*, April, 1954) conducted a follow-up study of patients with retrobulbar neuritis seen between 1937 and 1942 inclusive. Eighty-seven patients were studied. Of these, 28 developed evidence of multiple sclerosis; 26 were in the age group twenty to forty-four years. The authors conclude that an individual between the age of twenty and forty-five years who has an acute attack of retrobulbar neuritis has a 40% to 50% chance of having multiple sclerosis develop within ten to fifteen years. In patients under twenty years of age the chance of multiple sclerosis is less, and when it occurs after the age of forty-four years the chance is remote. Half of the patients between twenty and forty-four years who have an acute attack of retrobulbar neuritis may be expected to show other signs of multiple sclerosis within fifteen years.

On the Periphery.

GOLD.

Few will deny the power of gold. The language it speaks is understood by all. It opens doors and moves assemblies. It turns the hearts of some to double-dealing and dishonesty. Its beauty in ornament is matched by the ugly consequences of its worship. Gold is sought more ardently than wisdom. Some dig for it. Some glean it from the trampled fields of trade. Some steal it. It comes to some by inheritance, to others through the hazard of a lottery, seldom to those who are content to turn an honest penny. But whatever the pursuit, the appetite for it often becomes unruly and insatiable. Base motives then direct men's actions and inelegant thoughts disturb the tranquillity of their minds. Only the wise man (if such there be) remains unmoved at the sight of gold, for he knows the shining havoc it is likely to play in the lives of those who covet it. He, therefore, turns his face from the conceit of riches, and his aspirations to the pursuit of knowledge, little caring for the company of the money-changers in the temple or for those who poke their fingers into the pie-crust of pelf.

"Much have I travelled in the realms of gold", sang John Keats, not because he had camped on the diggings, but because he had read the classics, where myth and legend are so frequently tinctured with gold. Mythology features the Golden Age in which men lived without strife or labour on the earth's abundance. Æneas plucked a golden bough from a tree in the grove of Diana before descending into the nether world. Jason sailed with the Argonants to capture the golden fleece; and Jupiter, in order to perpetrate his invisible courtship of Danae, transformed himself into a golden shower. The crock of the leprechauns and the apples of the Hesperides were golden. The emerods of the Ark and the seven vials of God's wrath were of the same metal. Aaron erected a golden calf which encouraged the idolatry of his people; and everything which Midas touched turned into gold. Wherever one looks something golden springs into view, as if the imagination of man was gilded by the desire for opulence. The power and glory of kings are expressed in gold. The pirate's treasure, captured on the high seas, was likewise gold. It is among the resources of princes. Royal heads were clasped in its fillets. Nero built himself a house of gold. Henry VIII met the King of France on the Field of the Cloth of Gold. The rare and exalted of the earth have taken their food from golden plates. Golden goblets have toasted the birth of princelings. Beauty has admired its image in golden mirrors; and delicate and decadent limbs have been laved in golden baths. The worship of God is accomplished with golden vessels; and nearly every article pertaining to man's use or pleasure has at some time or other been made of gold. Even the close-stool of Beatrice d'Este, made for the easement of her stomach, was lined on the inside with virgin gold.

Gold entered the realm of medicine by another avenue than the fee. It had an honoured place in the pharmacopœia of magic. The older races revered its thaumaturgic powers. It was laid upon certain parts of the person with suitable incantations, or worn as a ring or an amulet. The cure, as it now is known, did not reside in the gold, but in the haphazardly sound principles of hygiene, as well as the faith which the charm inspired, and the persuasive powers of the doctor-priest who essayed the treatment. Its magic properties were prophylactic as well as curative. As a golden bell it was thought to banish the devil; and priests at one time had many such small articles stitched into their vestments so that when they moved the sound of the bells would protect them from evil spirits. Ignorance and gullibility gave gold its place among the chief magical remedies. The ancient Hindus had a most elaborate ceremony for curing jaundice, in the final stage of which red hairs of a bull, wrapped in gold leaf, were glued to the patient's side. The Greeks, to whom jaundice was the Golden Disease, naturally sought gold as a cure. The formula for this was to place a gold coin in a glass of wine and expose it to the stars for three nights. Three draughts of this mixture had to be imbibed daily until it was used up, by which time the jaundice was cured. Rumour does not relate how often the gold coin found a resting place in the pocket of the therapist while the ingenuous icteric was drinking his potion. But the sick are slow to doubt and remedies flourish among the unenlightened. So in southern Russia the jaundiced peasants were induced to wear golden bracelets; and in Germany yellow turnips, gold rings and golden coins were esteemed as valuable cures for jaundice. Gold was as powerful as prayer in mediæval medicine; and in the Renaissance its glory remained undiminished. In physic, according to Chaucer, gold was a cordial. It crept into the most desirable compounds, sharing a place with musk, amber and pearl in the famous confection given to the ailing ambassador of Queen Elizabeth. Royalty recognized its healing power, for, when suffering from typhoid, Louis XIV drank gold-leaf suspended in wine. Changing the usual practice of fee for service, when crowned heads ministered to the body's distempers by royal touch, they gave the patient whom they were about to cure a golden coin. This was frequently strung upon a white ribbon and hung round the patient's neck, where its therapeutic value was only less than the magic of that royal touch which in England was inaugurated by Edward the Confessor and died only with Queen Anne.

When medicine lay hidden in the morass of metaphysics and astrology the alchemists vainly strove to transform the baser metals into gold. In their search for the "philosopher's stone" they fruitlessly endeavoured to find an "elixir of life", and, with "potable gold", sought to cure illness. Gold had its place in the "doctrine of signatures"; and though it never lost its lustre, its therapeutic powers declined as science and controlled experiment gradually winnowed the grain from the chaff of modern treatment. In times anterior to the present gold salts have been prescribed in the treatment of epilepsy and hysteria. They were innocently exhibited to patients suffering from schizophrenia; and almost to this day were recommended in certain cases of phthisis and rheumatoid arthritis. In its long remedial history gold has passed through many phases, from the magic to the empiric; but in all this it has never quite lost, for either the patient or the doctor, its essential arcanum.

In this age of plastics and penicillin, in which the sovereign-case has followed the snuff-box into the past, the joyful tinkling of gold has given way to the odious crackling of bank-notes. Fiscal problems have relegated gold to the vaults of the Treasury. Its minted grace is seen no longer in the counting house where tellers tot up the mutable figures of the day's accountancy. The glitter is gone. Only the legal tender remains. Medical fees, though reckoned in guineas, are settled by paper tokens; and the bank draught takes the place of a handful of grateful gold. It was once the custom to gild the pill in order to render it more palatable; but no similar operation has been devised to make the doctor's account more acceptable. In other days when gold was current, before the guinea, the angel was the fee for medical service. Doctors looked hopefully for their fee—as they do now; but some were money-minded and avaricious and would, like Balaam's ass, refuse to budge before the angel appeared. As medicine became secularized and the profession was more highly organized, the fees increased. Large fortunes were made, particularly in the eighteenth century, in which many of those who carried a gold-headed cane as an emblem of their profession amassed considerable wealth. Quackery also reaped its unjust reward. But those whose fortunes were earned honestly gave their services freely to the poor and in many charitable ways helped to build up the humanitarian ideals of modern medicine. Gold has been the load-stone of the few, never the call of the many who practised medicine. Neither has the profession as a whole lacked compassion, or been unmindful of the reproach of pain. It is a consideration worthy of remark that most doctors live a life of unremitting toil, engrossed to the best of their ability in relieving sickness and implanting hope in the minds of those whom they cannot cure. Sharing an obscurity common to most men, yet valiant in sacrifice, determined in adversity and courageous in danger, the average doctor plies his profession with "nobler motives than a fee".

Gold is less in evidence today, since necessity became the mother of substitution; but it is still superlative in men's ideas. It symbolizes the varieties of excellence, the qualities of perfection. That which men most revere and that which is held in the highest rememberance may be linked with the golden epithet. The lives of the saints are preserved in the "Golden Legend"; the finest poems are enshrined in the "Golden Treasury"; and the acme of man's conduct is expressed in the golden rule. It is true that the "Golden Ass" is only an allegorical transformation, and that the bright balls of the pawnbroker have only the resemblance of gold. But the valiant man may still perform golden deeds, the orator may inspire with golden words and the generous man may possess a golden heart. If this was the case with all mankind, Milton's hope that "time will run back and fetch the age of gold" might soon be realized. But, alas, though the streets of heaven may be paved with gold, the highways of the earth are laid in stone, and set with the blood of men. Man's vision may reach the stars, but his feet are enmeshed in wickedness. Too often when gold glitters cupidity holds out its hands. Too often generosity

contracts when riches expand. The civilization that man has so laboriously wrought is built with bricks of greed and buttressed with racial hatreds. The monuments of his labour are blood-stained. His tenancy of the earth is in question. Technology may triumph and ingenuity lead to further invention, when the planet may become his plaything—and finally his tomb. The future holds no certainty. The sun breaks through, but the clouds are lowering. Meanwhile, perhaps, the only way to introduce another golden age may be to relinquish gold entirely.

R. S. ELLERY,
Melbourne.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

THE H. Douglas Stephens Memorial meeting of the Pædiatric Society of Victoria was held at the Royal Children's Hospital, Carlton, on June 9, 1954.

Intussusception.

DR. MURRAY CLARKE reviewed 164 cases of intussusception and DR. H. G. HILLER commented on radiological aspects of the subject including reduction with barium enema.

The papers will be published separately, but the following is a brief summary of Dr. Clarke's paper. The 164 cases of intussusception reviewed had occurred at the Royal Children's Hospital during the past five years. Dr. Clarke said that he had confirmed the observations of Morrison and Court, in Newcastle, and of Nelson, in Sydney, that the classical story of sudden attack followed by recurrent abdominal pain occurred only in a proportion of cases. Other combinations of symptoms were analysed.

During the past five years fourteen resections had been performed with two deaths, which compared well with other surgical figures. The cases which required resection were analysed in detail.

Dr. Clarke said that a compliment had been paid to Australian medical teaching by American authors when they attributed the Australian results to the alertness of practitioners in perceiving the condition and referring patients for surgical treatment. However, analysis showed that in an English series, 88% of those affected were admitted to hospital in forty-eight hours compared with 80% in Australia.

Six patients whose intussusception had been reduced at laparotomy or by barium enema during the past five years had died, with a resulting surgical mortality of 3·65%. Previous five-year mortality figures at the Royal Children's Hospital had been 12·4% in 1937 and 7·1% in 1948.

DR. R. SOUTHBY opened the discussion and commented that early diagnosis of the condition was the most important factor in reducing mortality. He said that any colicky pain with blood in the stool should be regarded as due to intussusception until proved otherwise.

DR. R. HOWARD commented on two features. He said that resection of large bowel carried a worse prognosis than resection of small bowel. In the Royal Children's Hospital cases ten of the resections were of large bowel, whilst in Jones's Newcastle series all but six were ileal resections. He thought that reduction by the use of barium was a forward step. Even if not successfully reduced, the intussusception was reduced to a large extent, and reduction could be completed through an oblique incision, which prevented the formation of adhesions if there was no big peritoneal scar. He did not agree that a barium enema should be used for diagnosis, but thought that it should be possible to palpate the tumour. The giving of a barium enema in such cases would be difficult except in a hospital such as the Royal Children's Hospital.

DR. W. FORSTER said that if he could not feel a tumour in a case of suspected intussusception, he administered a few drops of chloroform, thus facilitating greatly the palpation of the abdomen. A finger in the rectum would also often produce a small amount of blood and confirm the diagnosis. Dr. Forster asked whether one could get evidence of polypus from radiological examination.

DR. H. E. WILLIAMS remarked that he thought the use of barium as a means of diagnosis of abdominal pain was dangerous. Diagnosis could be made from a good clinical examination. He asked for the contraindications to the use of barium in reduction.

DR. F. D. STEPHENS suggested that manual intraabdominal reduction might cause trauma and referred to the case of Jones in which gangrenous patches were seen on the bowel. The use of barium might minimize that. Also occasionally at operation it was found that the intussusception had reduced itself. Such operations could be avoided if a barium enema was used first.

DR. B. HALLOWES also asked about the risks of barium enema. He said that in reviewing the published figures for different methods it seemed as though each man had become expert in his field and that it was the man behind the method that produced such good results.

DR. V. L. COLLINS congratulated Dr. Clarke on his presentation of the facts regarding cases of intussusception at the hospital, and said that now the question was how to improve them. It appeared that in the Newcastle series early diagnosis was the chief difference from the Royal Children's Hospital series, and he wondered what techniques were used in Newcastle to educate the general practitioners. He also asked whether there were any figures of the number of children in the series who had an anaphylactoid purpura. The type of intussusception associated with that condition simulated gastro-enteritis and was often ileo-ileal and therefore hard to diagnose by palpation or barium enema examination.

Dr. Hiller, in reply to Dr. Forster, said that it would just be chance whether barium enema examination showed up a polypus. In reply to Dr. H. E. Williams and Dr. B. Hallowes, he said that by cases of abdominal pain he had meant those thought to be intussusception, but in which no mass could be palpated. With regard to the contraindications to barium enema he said that overseas workers did not consider that there were any, but at the Royal Children's Hospital the case was assessed by the surgeon as to whether it was suitable. The degree of shock, dehydration and length of symptoms were all taken into account.

Dr. Clarke, in reply to Dr. V. L. Collins, said that in the series presented there were two cases of anaphylactoid purpura. The diagnosis was difficult, as the bleeding might be due to purpura and the lump a hæmatoma. He went on to say that the use of barium was not recommended generally for diagnosis, but he thought there were some cases in which the diagnosis need not have been delayed so long had barium been used. He agreed with Dr. Stephens that the use of a barium enema saved trauma to the bowel, as even reducible intussusceptions might have a split serosa from manipulation. He said that Dr. Hiller's figures were better than the average for barium reduction. The procedure needed a good radiologist, and it was necessary to guard against shock; so someone who understood that condition should be present. Children who were very ill, or were likely to need a resection or had abdominal distension, would not be submitted to administration of a barium enema at the Royal Children's Hospital.

Visual Loss in Childhood.

DR. R. S. HOOPER read a paper entitled "The Medical and Surgical Importance of Visual Loss in Childhood".

The paper will be published separately.

MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Theatre, New Medical School, Frome Road, Adelaide, on September 3, 1954.

The Glycolytic Effect in Bacterial Systems.

MR. A. L. HUNT read a paper on the glycolytic effect in bacterial systems. After describing some experiments on the nature of glucose stimulation of various bacterial systems, he went on to say that the presence of glucose was known to increase the rate of the following reactions in whole cells of the organism involved: the decarboxylation of malic acid to lactic acid, and of pyruvic acid to acetoin in *Lactobacillus arabinosus*; the deamination of aspartic acid in *Proteus vulgaris*, and the decomposition of formic acid in *Escherichia coli*. The pattern of glucose stimulation in all of those systems followed similar lines. The effect was produced only during concurrent glycolysis of the added sugar, without a lag period. No interval of stimulated activity existed after the sugar had been utilized. On

present biochemical knowledge a limited number of possible theories existed to explain the mechanism of this stimulation. They envisaged concepts of generation of energy (ATP) needed for the reaction, generation or regeneration of coenzymes, permeability effects, or a direct integration of glycolysis with the mechanism of the reaction itself. No evidence had been obtained as yet to confirm any of the first three possibilities in the system in *Lactobacillus arabinosus*, which converted pyruvic acid to acetoin and carbon dioxide. The mechanism of pyruvate decarboxylation in this organism had been found to involve an intermediate, acetolactic acid, formed by condensation of an "active aldehyde" unit from pyruvate, with another molecule of pyruvate. Acetolactic acid then lost carbon dioxide to give acetoin. The enzyme system involved in this new condensation reaction was being investigated. In cell-free extracts a competition for the "active aldehyde" unit existed between the condensing enzyme and oxygen, the former giving acetoin and the latter acetic acid. No stimulation by glucose could be obtained in cell-free extracts, but evidence was incomplete on whether the glycolytic sequence remained intact during disintegration of the cells. There was no evidence that glycolysis could affect pyruvic carboxylase, the first step in the reaction, nor did it stimulate acetolactate decarboxylation in whole cells. Thus, if a direct mechanism of glucose stimulation was to be postulated for the pyruvate system, the condensing reaction must be the one involved. It remained to be seen whether or not similar mechanisms existed for the widely differing systems in which the glucose effect could be observed.

Sucrose-Starch Interconversions in Plants.

In reporting a study of sucrose-starch interconversions in plants with radioactive sugars DR. L. H. MAY said that there could be little doubt that changes in starch and sucrose were interrelated in plants. Utilization of sucrose in starch synthesis could proceed by inversion followed by polymerization of the glucose moiety, in which case the fructose moiety must either accumulate or be utilized in respiration or other metabolic events. Alternatively, the whole sucrose molecule might be involved in the synthesis. Previous claims of a preferential utilization of fructose or glucose in starch synthesis and respiration had not been supported by adequate data. The fate of the fructose moiety of sucrose during starch synthesis was thus one of the outstanding problems, and the immediate aim of the work was to determine whether or not fructose contained in sucrose became incorporated into starch. Chemical analysis alone would provide information on this point only if more than half the sucrose transferred appeared as starch, and would in any case show only that an undefined amount of the fructose moiety had contributed to starch formation. By using radioactive sugars, however, the transformation of the moieties of sucrose could be followed in detail. Therefore, sucrose was synthesized in which either the glucose or fructose moiety was labelled and the two types were supplied to destarched leaf tissue. After suitable time intervals the redistribution of labelled carbon between starch, sugars and carbon dioxide was determined. The results had been compared with similar experiments in which invert sugar, with either the glucose or fructose labelled, was supplied. Results showed that fructose and glucose, free or combined in sucrose, were equally available for starch synthesis and as respiratory substrates. Hypotheses demanding preferential utilization had, therefore, proved untenable, and it was necessary to consider that the hexoses were drawn upon equally from a common metabolic pool.

The Ecological Web.

DR. H. G. ANDREWARTHA presented a paper entitled "Some Aspects of the Ecological Web".

Obituary.

GLEN HOWARD BURNELL.

WE have received from DR. F. L. WALL the following account of the late Dr. Glen Howard Burnell.

We will remember "Jimmy" Burnell with gratitude and respect. His medical career exemplifies the victory of a great mind and strong personality over a succession of severe bodily handicaps.

Born in 1890, Glen Howard Burnell's schooldays were finished at Prince Alfred College following a scholarship from the Adelaide High School. After a short term of clerical work, he realized that his vocation lay in medicine, and the initial years at the Adelaide Medical School were among the happiest in his life. At that time he was bodily fit, and he rowed intervarsity in the years from 1908 to 1911, the last crew winning the State, Intervarsity and Interstate Eights. While he was rowing, an haemoptysis occurred, herald of pulmonary tuberculosis. The following three years of enforced break from his medical course were spent in Central Australia, on the River Murray, and on a trip abroad. Returning in 1914, he topped his year at graduation in 1916. He then enlisted for military service, and was sent by the military authorities as resident medical officer to the Broken Hill Hospital. He later completed one voyage on the hospital ship *Karoola*, and was then discharged medically unfit for further military duties. He returned to Broken Hill Hospital, where he remained until 1921, becoming the Surgeon Superintendent. During this period he gained the M.D. (Adelaide) for a thesis on "The Control of Diphtheria", having eliminated this disease from the city of Broken Hill in the meantime.

His numerous early publications in THE MEDICAL JOURNAL OF AUSTRALIA bore evidence of breadth of mental scope and of academic leanings. They were as follows: "The Broken Hill Epidemic" (encephalitis or "X" disease) (1917 and 1918), "Two Cases of Pneumococcal Meningitis" (1918), "Treatment of Chorea" (1920), "Oral Administration of Ether as an Adjuvant to Open Ether" (1920).

After a short period of general practice in Broken Hill, Burnell was invited by the International Health Board of the Rockefeller Foundation to act as Director of the Hookworm Campaign in Australia. At that time he had the distinction of being the only member of the medical or technical staff who had never seen a hookworm! This appointment, his superintendence of the Broken Hill Hospital, and his initiation of their pathological laboratory were early evidence of his ability as an organizer. While administering the Hookworm Campaign in Queensland in 1922, he published a paper on "Hymenolepis Diminuta in Human Hosts".

Returning to Adelaide, Burnell next set up his plate in private practice in the house which was to become his permanent home. It was situated on the corner opposite the Wakefield Street Private Hospital, where he was later destined to carry out most of his private operations. His further story is that of a man of great personal drive and mental gifts pursuing the goal of a practising surgeon. His first honorary appointment was as surgeon to the outpatient department of the Adelaide Children's Hospital. For many years he was medical officer to the male night clinic at the Royal Adelaide Hospital. This practice in venereology laid the foundation of his particular interest and success in genito-urinary work. He obtained his Fellowship of the Royal College of Surgeons of Edinburgh in 1929, and in 1933 was appointed assistant surgeon at the Royal Adelaide Hospital. In 1943 he was appointed honorary surgeon at the Royal Adelaide Hospital, from which position he retired in 1950, at the age of sixty years. His particular surgical achievements and publications are dealt with elsewhere.

The writer knew "Jimmy" Burnell as a tower of strength in consultation. He was forthright in opinion, sometimes blunt, even gruff when provoked. He had a keen sense of humour, and he enjoyed a joke against himself. In his technical work one could detect honesty of purpose, courage and discretion, directing a highly intelligent brain and dexterous hands.

He shone also in spheres other than his surgical work. Lay people consulted him on purely business problems, and the medical fraternity made extensive use of his organizing and administrative ability. He was on the South Australian Branch Council of the British Medical Association and was Vice-President in 1949. Our interests were well served by him on the Council of the University of Adelaide from 1948. He was a member of the Council of the Institute of Medical and Veterinary Science, and a member of the Medical Board of South Australia and of the Nurses' Board of South Australia.

From 1949 he occupied the position of Chairman of Directors of the Wakefield Street Private Hospital. For many years he played golf, and was captain of the Mount Osmond Golf Club. His week-ends were often spent at Finniss, where for twenty years he owned and managed a large station. His pastoral interests were as enthralling to him as his medical ones, and equally successful. He loved knowledge for the sake of knowledge. He was proud of gaining high distinction on passing a correspondence course in wool-classing, undertaken when his practice was at its busiest. During the last two years, when limited to sedentary interests, he studied enthusiastically and became proficient in the Italian language. During his last five years two

major operations and coronary disease with complications greatly restricted his activities and tested his forbearance.

He was twice married. His first wife died in 1932, and in 1934 he married Dr. Mary Angel, who, with their three children, survives him. With her, a specialist anæsthetist, he worked in happy partnership on many a surgical problem. We mourn the loss of this brave outstanding man, to whose relatives we extend our deep sympathy.

SIR PHILIP MESSENT writes: There have been few more striking personalities among Adelaide's medical men than the late Glen Howard Burnell. A student of great promise, mentally and physically he was "set aside" for three years by illness. This setback but served to increase his fierce determination to reach the highest standards in his chosen profession. Life was never easy for him in his early years, and it is possible that his recurring struggles with difficulties did much to develop the character we admired and respected so greatly. At the conclusion of his career he was acknowledged to rank with the élite of Australian urologists. He achieved this distinction by unusual routes. In his early years he seemed equally interested in medicine, surgery and pathology, and he read widely and deeply in all three. This provided an unusually sound foundation on which to develop his surgical specialty. He conceived the idea of establishing a practice in venereal diseases with the ultimate intention of becoming a urologist. He explained his plans to several friends, but received little encouragement from them—they could see little chance of success. He thanked them and proceeded to prove that they were wrong. He did, in fact, develop a very large urological practice, and did much to pioneer this specialty in South Australia. He was always ready to accept new ideas, and to try fresh methods of surgical treatment. His technique in performing Mills's prostatectomy was excellent, and his results in a large series of cases were a great credit to him. One of his most engaging virtues was his keenness to help younger men, and his willingness to share with them the results of his large experience. "Jimmy" was an excellent teacher—partly because his energy and enthusiasm were infectious. No "loafer" could exist comfortably in his company. To whatever task he undertook, he gave his very best. No man could do more.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE MEDICAL BOARD.[1]

[From the *Australian Medical Journal*, June 1, 1847.]

IN redemption of the pledge given in our last number we now proceed to examine the constitution and conduct of "The New South Wales Medical Board". During the session of the Legislative Council of 1838 two acts were passed, one "to provide for the attendance of medical witnesses at Coroners Inquests and Inquiries held by Justices of the Peace", the other consequent on the first "to define the qualifications of Medical Witnesses", and empowering the Governor to appoint a Committee consisting of not less than three members of the medical profession under the title of the "New South Wales Medical Board" and to whom "any person deserving of being declared a legally qualified medical practitioner" for the purposes of the first named Act must submit his "degree diploma or other certificate or proof of his being so duly qualified". By two subsequent acts a branch Board was appointed for the Port Phillip district and the members of the Dublin Apothecaries Company were added to the qualified parties.

Now be it observed that the Act does not limit the Governor in his selection of the persons whom he may think proper to appoint as members of his Board and we find that the first Board consisted of five members, two of whom, Drs. Nicholson and Wallace, were private practitioners. The Board now—and we beg our readers to bear this in mind—consists of six members, five of whom either are or have been connected with the Government, the sixth being Dr. Nicholson who since his elevation to the chair of the Legislative Council has given up practice: so that we may fairly affirm that the private practitioners of the Colony are totally unrepresented in that Board. Let it also be observed that the President of the Board is Dr. Dawson and that two of its members—Messrs. P. Hill and Richardson—are official subordinates in his Department.

When the Board consists, as it ought to do, of one-third only of Government nominees and two-thirds of civil practitioners elected by the profession then may we expect its influence to be used and its power exerted to benefit the general body, till then it will only be the instrument of their degradation and of the grasping avarice and monopolizing ambition of an individual. "Coming events cast their shadows before them." Let not our warning be despised.

Since the foregoing was written we have been reminded by reference to the Report of the Select Committee on the Lunatic Asylum of another piece of patronage and emolument that it is proposed by that body to confer on Dr. Dawson in addition to those he already enjoys. "Some are born great, some achieve greatness, and some have greatness thrust upon them." To which of these classes Dr. Dawson belongs we leave our readers each to judge for himself. Our vocation is to protest in the name of the profession in the strongest language we can command against this piling of honours and emoluments upon the head of one individual as though there were no others equally deserving of them. It is a gross insult to our body. What has Dr. Dawson ever done to advance medical science? Either the employments he already has are sufficient to occupy his time, or he is much overpaid for them and ought if he *must* have the honour to be satisfied with that: but if a new salaried office is to be erected, let it be conferred on someone, who while he proves himself able to perform the duties required, may have some additional inducement to perform them well, from the solid advantage he acquires by the welcome addition of the salary to his present precarious and perhaps insufficient income.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

MEDICAL DEFENCE UNION INSURANCE IN PARTNERSHIP PRACTICE.

SIR: Questions have been frequently asked by members of the New South Wales Medical Defence Union, Limited, who are engaged in partnership practice, concerning their position where legal action may be taken against one member of the partnership and in addition the partnership as a whole is joined in the action.

It is often pointed out: (*a*) that only one member was actually concerned in the treatment of the patient who was never seen professionally by any of the others; (*b*) whilst one member actually carried out the treatment, other members of the partnership did, at times, examine the patient.

To clarify the matter beyond doubt we have been in touch with our overseas reinsurers through our solicitors.

We are now assured by the reinsurers that any member of the Medical Defence Union of New South Wales is covered for the full amount of his indemnity in any action which may be taken against him as the individual directly responsible or merely as a member of a partnership (who may not have seen the patient professionally, or may have done so only incidentally), where the claim arises out of the legitimate professional practice of the partnership.

Yours, etc.,

B. T. EDYE, President,
The New South Wales Medical Defence Union, Limited.

185 Macquarie Street,
Sydney,
October 14, 1954.

AN APPEAL.

SIR: On August 30, 1954, at approximately 6.30 p.m. on Monday my son was involved in a fatal accident at the corner of Liverpool Road and Grosvenor Crescent, Ashfield. He was driving a panel van and was struck by a car travelling in the direction of Ashfield. The force of the impact on the near side rear wheel caused the vehicle to spin and it rolled over on its side, pinning my son beneath it.

While he was trapped beneath this he was attended by a passing doctor of whose name we are unaware. I would be very grateful if this doctor could get in touch with me either by 'phone or letter.

Yours, etc.,

8 Richmond Avenue, Ashfield. (Telephone UA 8102.)
October 18, 1954.

W. E. BROWN.

INDUCTION OF LABOUR.

SIR: The interesting article in your journal of the second instant provokes a few remarks from me, which I crave a little space in your journal to express.

Having taken my first steps in midwifery as it was then known some forty-eight years ago, I must regard myself as old-fashioned, though in common with many others I do try to keep abreast of modern trends and changes. In those far-off days when one's hair was lush and black with the vigour of enthusing youth, it used to be taught that certain criteria had to be satisfied before induction could be permitted—namely: (i) The head must be presenting. (ii) The head must be sinking into the brim of the pelvis. (iii) The cervix must be soft and almost effaced. (iv) The os must be dilated enough to admit at least one finger.

Looking at modern teaching, Sir, one does not see any difference—*vide* the *Practitioner*, special midwifery number, February, 1951 (a current and popular journal is quoted in preference to a text-book). On page 546 of your above-quoted journal, Dr. Bellingham promotes and provokes a "ripe" cervix by performing a medical induction which seems like placing "the cart before the horse". A second point is his practice of repeating induction to get a "ripe" cervix. This is quite contrary to the teaching in my days, and it was always looked on with horror and regarded as very dangerous and meddlesome midwifery. A mere suggestion of it in those days would have deprived one of a passport at an examination.

The third and last point, Sir, which gives me some difficulty is inducing labour at thirty-five to thirty-six weeks in Rh-negative women with agglutinins, more so if there is a history of previous erythroblastosis (see page 547).

I thought that induction had no place in these cases, and only an exceptionally high titre of agglutinins might make one consider it, each pregnancy behaving in its own way. Trying to save the infant's life in such a case may lead to the loss of both mother and babe.

I would be very grateful if someone in real academic authority, say the Professor of Obstetrics, University of Queensland, will give us mere general practitioners the benefit of his views and tell us what he himself teaches.

I have no desire to be controversial, Sir; I am only somewhat puzzled and am looking for guidance.

Yours, etc.,

District Hospital, Boulia, Queensland.
October 11, 1954.

B. J. BOUCHÉ, M.R.C.S., L.R.C.P.

TOBACCO SMOKING AND LUNG CANCER.

SIR: If the association between smoking (especially heavy smoking) and lung cancer is real, it occurs to me that there is one question that has not been asked and that I think should be asked. Why do these people smoke at all, and some of them smoke so much? Perhaps our psychologist colleagues might be able to answer this.

Another interesting question is this: Why do pipe smokers have a much lower incidence of lung cancer than cigarette smokers and a higher incidence of cancer of the buccal cavity? We all remember the time-honoured phrase about one of the possible causes of cancer of the lips and tongue: "The irritation of a short clay pipe."

Why does the pipe smoker suck that pipe, even, sometimes, when it is empty? And why does the cigarette smoker inhale the fumes into his lungs?

The incidence of lung cancer rises steeply and progressively from the age of forty-five years onwards (*Brit. M. J.*, June 26, 1954), that is, during the period when sexual potency is on the wane.

Might it be that the pipe smoker is unconsciously gratifying an oral eroticism? And in the case of the cigarette smoker, is he filling a hollow organ, the lung, instead of another hollow organ he can no longer fill, the uterus?

These considerations may seem far-fetched. Nevertheless, my original question remains: Why do these people smoke at all, and most of them (*Brit. M. J., loco citato*) smoke so much?

Yours, etc.,

British Medical Association House,
135 Macquarie Street,
Sydney.
October 15, 1954.

ARTHUR D'OMBRAIN.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Bega.

The Post-Graduate Committee in Medicine, in conjunction with the Far South Coast and Tablelands Medical Association, will hold the following week-end course at Bega on Saturday and Sunday, November 20 and 21, 1954:

Saturday, November 20: 2 p.m., Registration; 2.15 p.m., "Hæmorrhoids, Anal Fissure and Fistula", Dr. R. M. Rawle; 3.45 p.m., "Bronchitis and Bronchiectasis", Dr. H. M. Windsor.

Sunday, November 21: 9.30 a.m., "Modern Trends in Pædiatrics", Dr. Bryan A. Curtin; 10.30 a.m., "Some Interesting Gastro-Intestinal Lesions", Dr. R. M. Rawle; 11.30 a.m., "Surgical Aspects of Mitral Stenosis", Dr. H. M. Windsor; 2 p.m., "Some Common Problems in Pædiatrics", Dr. Bryan A. Curtin.

The fee for attendance at the course will be £3 3s. Those wishing to attend are requested to notify Dr. E. C. Blomfield, Honorary Secretary, Far South Coast and Tablelands Medical Association, 104 Gipps Street, Bega, New South Wales.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 56 and 57, of September 16 and 23, 1954.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Termination of Appointment.—The appointment of Brian Hayley Webb as Surgeon Lieutenant (for Short Service) is terminated, dated 13th August, 1954.

Appointment.—Michael Joseph Lydon is appointed Surgeon Lieutenant-Commander (for Short Service), dated 26th January, 1954.

Loan to the Royal Navy for Service and Training.—The appointment of Michael Joseph Lydon as Surgeon Lieutenant (for Short Service) is terminated, dated 25th January, 1954.

Citizen Naval Forces of the Commonwealth.

Corrigendum.

With reference to Executive Minute No. 14—notice of which appeared in *Commonwealth Gazette* No. 17 of 11th March, 1954—that portion relating to the promotion of Surgeon Lieutenant (for Short Service) Michael Joseph Lydon is cancelled.—(Ex. Min. No. 85—Approved 14th September, 1954.)

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/39083 Captain R. Cantamessa is seconded whilst undergoing post-graduate studies in the United Kingdom, 1st February, 1954. 1/39130 Captain P. J. F. Grant ceases to be seconded for post-graduate studies in the United Kingdom, 3rd August, 1954. 1/39175 Honorary Captain H. H. Moy is appointed

from the Reserve of Officers, and to be Captain (provisionally), 23rd July, 1954.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: Captains 2/79109 R. L. Manning, 2/101562 J. V. Roche and 2/127883 J. T. Dunn.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/101018 Captain W. N. Sloan is seconded whilst undergoing postgraduate studies in the United Kingdom, 15th May, 1954.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—5/32269 Honorary Captain J. B. O'Brien is appointed from the Reserve of Officers, and to be Captain (provisionally), 30th July, 1954. To be Captain (provisionally), 16th August, 1954: 5/21605 Maxwell Clive Canning. To be Temporary Major, 16th August, 1954: 5/21578 Captain C. Stuart, M.C.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).

The Short Service Commission granted to 3/40123 Captain (Temporary Major) A. P. Hanway is extended until 30th September, 1955.

ROYAL AUSTRALIAN AIR FORCE.

Active Citizen Air Force.

Medical Branch.

No. 24 (City of Adelaide) Squadron.—Flight Lieutenant C. D. Swaine (041135) is granted the acting rank of Squadron Leader, 31st July, 1954.

No. 25 (City of Perth) Squadron.—Flight Lieutenant J. R. H. Watson (051326) is transferred from the Reserve, 31st October, 1953.

The Royal Australasian College of Physicians.

RESEARCH FUND.

THE ROYAL AUSTRALASIAN COLLEGE OF PHYSICIANS invites medical practitioners to submit applications for grants from the Research Fund of the College before November 30, 1954. Grants may be made for research fellowships tenable in Australasia or overseas, or as grants in aid in approved circumstances. Applications should be made on the prescribed form, which may be obtained from the Honorary Secretary of the College at 145 Macquarie Street, Sydney.

ADMISSIONS TO MEMBERSHIP.

THE following, having satisfied the Board of Censors in Australia, were admitted to membership of The Royal Australasian College of Physicians by the President in October, 1954: B. S. Hartnett, A. P. Millar and J. D. Murphy, of New South Wales; I. H. W. Anderson, J. R. E. Fraser, G. J. Groves, W. H. Kitchen, Graeme Larkins, D. H. Meyers and M. G. Whiteside, of Victoria; C. H. Campbell, of Queensland; T. S. Kirkland, of Tasmania.

Notice.

VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION: SECTION OF PREVENTIVE MEDICINE.

THE last meeting for 1954 of the Section of Preventive Medicine of the Victorian Branch of the British Medical Association will be held in the Medical Society Hall, 426 Albert Street, East Melbourne, at 4.30 p.m. on Thursday, November 11, 1954. Dr. H. McLorinan, Medical Superin-

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 9, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	5(3)	3(1)	2	..	2(2)	..	..	2	14
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	1	..	1	..	..	..	..	..	2
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	2(1)	..	..	1(1)	..	..	..	3
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1	8(6)	4(4)	..	1(1)	..	..	6	20
Diphtheria	..	5(4)	..	1	..	..	..	..	6
Dysentery (Bacillary)	..	1(1)	1(1)	..	..	..	..	..	2
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	33(15)	56(37)	..	..	1	..	..	2	92
Lead Poisoning	..	..	..	..	1	..	..	..	1
Leprosy	..	1(1)	..	..	10	..	..	..	11
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(2)	5(3)	..	..	..	1	1	..	10
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	1	..	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2(1)	11(5)	3	5(5)	..	..	..	..	21
Puerperal Fever	..	..	..	..	..	..	..	1	1
Rubella	..	17(9)	..	..	17(12)	..	..	3	37
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	12(8)	33(20)	7(6)	5(5)	..	..	..	..	57
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	12	..	1	..	13
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	20(15)	20(12)	9(5)	6(4)	4(3)	1	4	..	64
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	1	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

tendent of the Fairfield Hospital, will give an address entitled "The Changing Picture of Infectious Diseases". All members of the Branch are invited to be present.

SUMMER CAMPS FOR DIABETIC CHILDREN.

MEDICAL PRACTITIONERS who have under their care diabetic children aged between six and thirteen years are advised that the annual camp for diabetic children is to be held at the Fairbridge Farm School, Molong, for two weeks commencing on January 8, 1955. The children are under the care of a trained nurse and a dietitian. Organized activities are a feature of the camp. The expenses per child for the two weeks amount to £10, and parents are asked to pay only as much of this as they can afford. Further information and application forms are obtainable from the Secretary, Miss N. Kirk, c.o. Dietitian's Office, Royal North Shore Hospital of Sydney, Crows Nest.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1953, as duly qualified medical practitioners: Jelihovsky, Tatiana, M.B., B.S., 1953 (Univ. Hong Kong); O'Reilly, Cyril Francis, M.B., B.Ch., 1936 (Q.U., Belfast), D.P.M. (Dublin), 1940; Watson, Jean Patricia, M.B., Ch.B., 1951 (Univ. Aberdeen).

The following additional qualifications have been registered: Kerridge, John Charters (M.B., B.S., 1942, Univ. Sydney)—D.P.M. (Univ. London), 1951; De Monchaux, Robert Joseph (M.B., B.S., 1946, Univ. Sydney)—M.R.A.C.P., 1950; Opie, Richard Cunynghame (M.B., 1941, Univ. Sydney) —M.S., 1952 (Univ. Sydney); Rennie, Harry Maynard (M.B., B.S., 1928, Univ. Sydney, M.R.C.P., London, 1942, F.R.A.C.P., 1946)—F.R.C.P., London, 1954.

Suspension from Practice.—Carr, Kenneth Allan, Urana, M.B., B.S., 1950 (Univ. Sydney), has been suspended from practice for a period of three months from October 1, 1954.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners: O'Halloran, John, M.B., B.S., 1954 (Univ. Sydney); Whitehouse, Harry, L.M.S.S.A. (London), 1953, M.R.C.S. (England), L.R.C.P. (London), 1954.

The following additional qualifications have been registered: Aitken, Ronald Edward, F.R.C.S. (England), 1950; Lee, John Francis, F.R.C.S. (England), 1954.

Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Dobbie, John Alastair, M.B., B.S., 1954 (Univ. Sydney); Egan, John Desmond, M.B., B.S., 1954 (Univ. Sydney); Killingback, Marcus James, M.B., B.S., 1954 (Univ. Sydney); Stange, Fay Joan, M.B., B.S., 1954 (Univ. Sydney); Fisher, Robin Gerard Wyndham, M.B., B.S., 1953 (Univ. Sydney); Wallace, Gilbert Hugh Murray, M.B., B.S., 1952 (Univ. Sydney); Fetterplace, Reginald John, M.B., B.S., 1950 (Univ. Sydney); de Wilde, Frans Willem, registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953; Roston, Siegfried, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1953.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Harris, David John, M.B., B.S., 1952 (Univ. Adelaide); Mackie, Dean Graham, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953); Richards, James Ross, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953); Dow, Lorna Ruth, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953).

Medical Appointments.

Dr. J. S. L. Carew has been appointed government medical officer at Herberton, Queensland.

Dr. I. B. Jose has been appointed a member of the Advisory Committee pursuant to the *Hospitals Act*, 1934-1951, of South Australia, as the nominee of the Council of The University of Adelaide for the balance of the term for which the late Dr. G. H. Burnell was appointed.

Deaths.

THE following death has been announced:

GIBLIN.—William Eric Giblin, on October 20, 1954, at Sydney.

Diary for the Month.

Nov. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 10.—Victorian Branch, B.M.A.: Clinical Meeting.
Nov. 12.—Queensland Branch, B.M.A.: Council Meeting.
Nov. 15.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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